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TOXICITY AND REACTIONS CAUSED BY ARSPHENAMIN AND NEO-ARSPHENAMIN

THE EFFECT OF ORGANIC COMPOUNDS OF ARSENIC, MERCURY AND BISMUTH ON THE KIDNEYS OF ANIMALS, JUDGED BY THE NONPROTEIN NITROGEN AND UREA CONTENT OF THE BLOOD *

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The purpose of the investigation the results of which are described in this paper, was to discover what symptomatic effects, if any, various doses of arsphenamin, neo-arsphenamin and organic compounds of mercury and bismuth had on the renal function of animals.

One phase of this problem has already been amply covered by the researches of Drs. J. A. Kolmer and B. Lucke,[1] who made a study of the histologic changes produced experimentally in rabbits by the arsenicals and various compounds of mercury named above. In their necropsy examinations, animals treated with one large dose of arsphenamin showed "severe vascular and tissue alterations, particularly in the liver, kidney, suprarenals and spleen"; those injected with a "single . . . massive dose of neo-arsphenamin produced vascular injury, cellular degenerations and necrosis similar to but less severe than those produced by arsphenamin," while all mercurials employed caused tissue changes in all the organs examined.

Now it has been shown within the last decade that impairment of the renal function almost invariably results in an increase of the nonprotein and urea nitrogen, and, if the impairment is severe enough, also of the creatinin content of the blood. Thus, Farr and Austin[2] have found, after a long series of blood examinations of hospital patients, that chronic nephritis in general and uremia in particular are "almost always accompanied by an increase of nonprotein nitrogen in the blood."

* From the Dermatological Research Laboratories, Philadelphia.

1. Kolmer, J. A., and Lucke, B.: Study of Histologic Changes Produced Experimentally in Rabbits by Arsphenamin, Arch. Dermat. & Syph. **3**:483 (April) 1921.

2. Farr and Austin: J. Exper. Med. **18**:228, 1913.

The following year Tileston and Comfort,[3] conducting similar experiments, came to the conclusion that uremia always, and chronic nephritis usually, causes an abnormal amount of nonprotein nitrogen and urea to be retained in the blood, although they add that a similar rise has usually been observed in the blood of patients suffering from acute intestinal obstruction, acute lobar pneumonia and occasionally in syphilitic persons. Mosenthal and Miller,[4] however, go further, and claim, as a result of their investigations, that "to a certain extent, therefore, the increased percentage of urea nitrogen may be regarded as a sign of renal insufficiency, even when total non-protein nitrogen is within normal limits."

An increase of the creatinin content of the blood is an even surer sign of renal disturbance as the kidneys have a remarkable power for eliminating this waste product, which is not retained until they have sustained considerably injury.

From the foregoing it is evident that a high nonprotein nitrogen and urea content of the blood is closely, although not absolutely, associated with an impairment of the renal function, and a disturbance of the latter in healthy animals, after injections of arsphenamin or neoarsphenamin, would unmistakably be due to the destructive effect of these drugs on the kidneys.

It will be interesting to review briefly the studies made on the effect of intravenous injections of therapeutic doses of arsphenamin and neoarsphenamin on the kidneys of syphilitic persons. With the exception of Weiss and Corson, the following authors limited their work to a single chemical analysis of the blood (and also of the urine) of the patient after the administration of a course of arsphenamin injections, to discover whether or not the kidneys have in any way been injured. In this manner Rowntree, Marshall and Chesney [5] observed a high nonprotein nitrogen of the blood (150 mg. in 100 c.c. of blood) in a case of tabes dorsalis with acute and chronic nephritis, in which death resulted from arsphenamin poisoning.

Rappleye,[6] after studying several groups of patients, concluded that arsphenamin has practically no deleterious effect on the kidneys when their function is good at the outset.

Elliot and Todd [7] carried on investigations along similar lines and obtained similar results. No marked increase in the amount of non-

3. Tileston, W., and Comfort, C. W.: Total Non-Protein Nitrogen and the Urea of the Blood in Health and in Disease as Estimated by Folin's Methods, Arch. Int. Med. **14**:620 (Nov.) 1914.

4. Mosenthal and Miller: J. Urol. **1**:75, 1917.

5. Marshall and Chesney: Trans. Assn. of Am. Phys. **29**:586, 1914.

6. Rappleye: J. Lab. & Clin. Med. **4**:630, 1919.

7. Elliot, J. A., and Todd, L. C.: Effects of Arsphenamin on Renal Function in Syphilitic Patients, Arch. Dermat. & Syph. **2**:699 (Dec.) 1920.

protein nitrogen or urea was found in the blood. In one case of acute syphilitic nephritis with high blood urea they even observed a rapid decrease in the latter under arsphenamin treatment.

The works of Bailey and MacKay,[8] and of Anderson [9] which report nonprotein nitrogen findings in syphilitic patients somewhat higher than normal do not very well fit into this review, as their patients had also received mercury treatments, the injurious effect of which on the kidneys is well known.

The study of Weiss and Corson [10] is of greater significance, because their work was comparative and, therefore, more illuminating. These authors focused their attention on five hospital patients with tertiary syphilis with varying degrees of optic atrophy, whose blood was examined at various times both before and after treatment. These chemical analyses showed "small but definite increases in the nonprotein nitrogen of the blood (2.5 mg. per 100 c.c.) . . . three hours after practically every intravenous injection of 0.6 gm. doses of arsphenamin. . . . The increases in urea nitrogen were not always parallel to those in the nonprotein nitrogen, and often were absent or exceeded them." They also add the significant fact that "in no case was the final total nonprotein or urea nitrogen of the patient, when discharged (after one, two or three 0.6 gm. doses of arsphenamin), any higher than when admitted. On the contrary many reductions were noted."

With this preliminary review in mind, we may now approach our own contribution to this problem. In order to make the work more uniform so as to enable us to arrive at definite conclusions, the blood of the rabbits was examined for total nonprotein nitrogen, urea nitrogen and creatinin once before the administration of the drug and at various periods after it. The plan of the arsenical injections was to give one animal one massive dose approaching the maximum tolerated dose for rats; to another animal one large dose (about one half of the maximum tolerated dose); then to a third the usual therapeutic dose, and finally one rabbit was to receive twelve therapeutic doses, at intervals of three or four days. The work was done in duplicate and frequently in triplicate to insure reliable results. The animals were also kept under uniform conditions, and on an approximately constant diet. The results are shown in the tables.

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8. Bailey, C. V., and MacKay, A.: Toxic Jaundice in Patients Under Antisyphilitic Treatment, Arch. Int. Med. **25**:628 (June) 1920.

9. Anderson: Am. J. Med. Sc. **162**:80, 1921.

10. Weiss, C., and Corson, A. C.: Chemical Studies of the Blood and Urine of Syphilitic Patients Under Arsphenamin Treatment, Arch. Int. Med. **29**:428 (April) 1922.

One hundred milligrams per kilogram is generally considered as the maximum tolerated dose, though greater amounts, even as high as 160 mg. per kilogram, are occasionally tolerated by white rats. Parenthetically we may add that in these studies we have found that rabbits did not tolerate relatively as high doses of arsphenamin as did white rats.

These two experiments clearly show the injurious effects of massive doses of the drug. Rabbit 1 showed a fairly rapid rise of the nonprotein and urea nitrogen of the blood, and died on the tenth day after injection, following a severe diarrhea. Necroscopic examination showed nephritic kidneys.

In Rabbit 2 the results became apparent much sooner, probably due to the higher toxicity of this particular lot of the drug. The rise in the nonprotein and urea nitrogen and even in the creatinin of the blood was precipitous, the animal dying seven hours after injection, following severe diarrhea, a reaction so frequent after the administration of the drug to human beings.

TABLE 1.—*The Intravenous Injection of One Massive Dose of Arsphenamin into Rabbits*

Experiment	Dosage Mg. per Kilogram	Weight in Grams	Time when Blood Was Taken	Mg. per 100 C.c. of Blood		
				Nonprotein Nitrogen	Urea Nitrogen	Creatinin
1	100	1,970	Before injection	34	18	1.5
		1,900	7 hours after injection	40	21	1.8
		1,870	6 days after injection	60	48	1.7
2	100	1,940	Before injection	37	13	1.6
			3 hours after injection	113	78	2.4

The results shown in Table 2 are interesting. The quantity of the compound administered to the rabbits was five times that of the usual therapeutic dose. Yet all of them not only survived, but recovered completely within from ten to sixteen days. There can be no doubt, however, as to the injury to the kidneys caused even by this dose. The nonprotein nitrogen, urea nitrogen and creatinin content of the blood rose immediately after injection and reached a crisis on the third day, when the content of each of the three waste products in the blood was the highest, the differences in the actual quantities found in each of the three experiments probably being due to the varying toxicities of each of the three samples of the drug used, although the individual idiosyncrasies of the animals must also be taken into account. After the third day the condition of the kidneys began to improve, and the recovery was completed within a week or two.

Table 3 demonstrates the effect of a small dose of arsphenamin on the kidneys. The largest single dose usually employed in the therapy of human syphilis is 0.6 gm., and as the weight of the average person is

assumed to be about 60 kilograms, the dose given in the practical treatment of syphilis is 10 mg. per kilogram. From Table 3 we may conclude, therefore, that single therapeutic doses of the drug result in no apparent disturbance of the renal function.

These results, however, could not be assumed to represent the approximate final effect of the medicament on the human kidneys, as the patient usually receives his treatment in courses of from six to

TABLE 2.—*The Intravenous Injection of One Large Dose of Arsphenamin into Rabbits*

Experiment	Dosage Mg. per Kilogram	Weight in Grams	Time when Blood Was Taken	Mg. per 100 C.c. of Blood		
				Nonprotein Nitrogen	Urea Nitrogen	Creatinin
2	50	3,650	Before injection	35	17	1.8
		3,690	2½ hours after injection	47	21	1.5
		3,565	3 days after injection	95	71	3.0
		3,440	7 days after injection	53	27	—
		3,600	10 days after injection	50	25	1.6
		3,530	14 days after injection	37	18	—
4	50	2,850	Before injection	40	18	2.0
		2,800	2 hours after injection	46	23	—
		2,670	3 days after injection	71	59	3.0
		2,650	6 days after injection	69	40	2.8
		2,700	9 days after injection	59	31	2.5
		2,700	13 days after injection	48	25	—
		2,720	16 days after injection	42	19	1.9
5	50	2,330	Before injection	34	16	—
		2,200	2 hours after injection	44	26	1.6
		2,055	3 days after injection	59	34	1.9
		2,260	6 days after injection	46	24	1.7
		2,281	10 days after injection	37	16	—

TABLE 3.—*The Intravenous Injection of One Therapeutic Dose of Arsphenamin into Rabbits*

Experiment	Dosage Mg. per Kilogram	Weight in Grams	Time when Blood Was Taken	Mg. per 100 C.c. of Blood		
				Nonprotein Nitrogen	Urea Nitrogen	Creatinin
6	10	1,690	Before injection	35	16	1.4
		1,815	2.5 hours after injection	35	15	1.5
		1,840	3 days after injection	33	15	1.5
7	10	2,100	Before injection	34	16	1.5
		2,205	2.5 hours after injection	34	17	1.5
		2,200	3 days after injection	36	17	1.4
8	10	2,030	Before injection	36	17	1.5
		2,010	1 day after injection	35	14	1.4
		2,040	3 days after injection	38	18	1.5

twelve injections at intervals of one week or oftener. We, therefore, injected into two rabbits therapeutic doses of arsphenamin twice a week, at intervals of three and four days, for six weeks, making a total of twelve doses, the combined weight of which exceeded the maximum tolerated dose for rats, which was found to be 100 mg. per kilogram.

The results, which are given in Table 4 were satisfactory. Not only did the renal function of the animals remain unaffected by the repeated

injections of the drug, but the rabbits even gained weight, especially Rabbit 9.

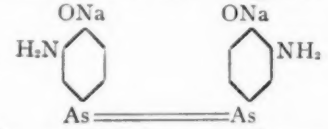
To sum up our results: We may affirm that single massive doses of alkalinized arsphenamin produce severe nephritis often followed by death; that single large doses of the drug produce acute nephritis, which generally clears up within from ten to sixteen days, and that repeated therapeutic doses do not disturb the renal function at all.

We were now interested to discover what part of the arsphenamin molecule did the damage when injected in massive or large doses; in other words, we wanted to know whether it was the arsenic or the

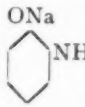
TABLE 4.—*The Intravenous Injection of Multiple Therapeutic Doses of Arsphenamin into Rabbits*

Experiment	Dosage Mg. per Kilogram	Weight in Grams	Time when Blood Was Taken	Mg. per 100 C.c. of Blood		
				Nonprotein Nitrogen	Urea Nitrogen	Creatinin
9	10	2,175	Before injection	38	16	1.7
		2,200	2.5 hours after first injection	33	14	1.5
		2,200	3 days after first injection	36	16	1.6
		2,170	1 day after second injection	35	14	1.6
		2,190	1 day after third injection	32	13	—
		2,150	1 day after fourth injection	34	16	1.5
		2,200	1 day after fifth injection	32	13	—
		2,410	1 day after sixth injection	35	16	1.5
		2,300	1 day after seventh injection	34	16	—
		2,425	1 day after eighth injection	38	17	—
		2,500	1 day after ninth injection	35	17	1.6
		2,515	1 day after tenth injection	38	18	—
		2,410	1 day after eleventh injection	—	18	1.6
		2,440	1 day after twelfth injection	37	17	1.5
10	10	2,275	Before injection	40	18	1.5
		2,340	1 day after first injection	41	19	1.5
		2,260	1 day after second injection	38	18	1.4
		2,040	1 day after third injection	38	16	—
		2,070	1 day after fourth injection	39	18	1.6
		2,145	1 day after fifth injection	35	14	—
		2,285	1 day after sixth injection	36	17	1.5
		2,250	1 day after seventh injection	37	18	—
		2,325	1 day after eighth injection	40	20	—
		2,350	1 day after ninth injection	38	19	1.5
		2,250	1 day after tenth injection	32	14	—
		2,240	1 day after eleventh injection	35	17	1.5
		2,300	1 day after twelfth injection	36	18	1.4

remainder of the molecule that produced renal disturbance. A glance at the formula of alkalinized arsphenamin



will make it evident that the molecule may be considered as a condensation of two molecules of sodium-o-aminophenolate



each containing in addition a trivalent arsenic atom in the position para to the hydroxyl. We, therefore, injected three normal rabbits with an

alkalinized solution of 100, 150 and 200 mg. per kilogram, respectively, of ortho-aminophenol, the results being given in Table 5.

A brief study of Table 5 in the light of what has been said above, will make it clear that it is the arsenic, and nothing but the arsenic, in the arsphenamin molecule that is responsible for the injuries to the kidneys, for a compound identical with the remaining part of the arsphenamin molecule produces slight or no renal disturbance even when injected in doses twice as large as the maximum tolerated dose of arsphenamin.

However, the results of Experiments 11, 12 and 13 have shown us another phase of the action of arsphenamin on the animal body. In the reactions, chills and convulsions that followed the injection of sodium-

TABLE 5.—*The Intravenous Injection of Alkalinized Solutions of Ortho-Aminophenol into Rabbits*

Experiment	Dosage Mg. per Kilogram	Weight in Grams	Time when Blood Was Taken	Mg. per 100 C.c. of Blood: Nonprotein Nitrogen	Urea Nitrogen	Creatinin
11	100*	2,700	Before injection	37	17	1.5
		2,850	2.5 hours after injection	40	18	—
		2,900	3 days after injection	44	18	—
		2,920	7 days after injection	42	18	1.4
12	150†	2,350	Before injection	41	18	1.6
		2,300	2.5 hours after injection	41	17	—
		2,400	3 days after injection	40	15	1.5
		2,390	7 days after injection	40	19	—
13	200‡	1,715	Before injection	44	20	1.7
		1,795	2.5 hours after injection	44	22	—
		1,880	3 days after injection	40	18	1.5

* Severe reaction; recovered within fifteen minutes.
† Violent reaction; convulsions; recovered within thirty minutes.
‡ Violent reaction; chills, convulsions; recovered within one hour.

ortho-aminophenolate, which became more violent as the dose increased, one recognizes the unwelcome manifestations which frequently appear after the administration of arsphenamin to syphilitic persons. There is, then, a possibility that the cause of at least some of the reactions following arsphenamin lies in the aminophenol radical. This highly important clue will be investigated further by the authors.

Thus far we have been dealing with arsphenamin. Neo-arsphenamin now finds increasing favor in the eyes of syphilologists, and is now the more widely used of the two. We, therefore, thought it advisable to carry out a series of similar experiments with neo-arsphenamin.

The maximum tolerated dose of neo-arsphenamin for white rats is generally considered to be 200 mg. per kilogram of body weight, but products can be and have been made which were tolerated in doses as high as 300 and even 400 mg. per kilogram. The drugs injected into

Rabbits 14 and 15 were tolerated by white rats in doses of 300 mg. per kilogram, while that employed on Rabbit 16 passed at 400 mg. per kilogram.

A glance at the records of Rabbits 14 and 15 will bring to mind the assertion we made a few pages earlier, namely, that rabbits show a lower tolerance for these drugs than do white rats. The dose of 300 mg. per kilogram which was easily tolerated by the latter produced a severe diarrhea in Rabbit 14 one hour after injection. Four hours after injection the values for nonprotein nitrogen, urea nitrogen and creatinin rose, resulting in death two hours later.

Nor did Rabbit 15 fare much better. Because of the smaller dose the results were more tardy in their appearance, but on the fifth day the

TABLE 6.—*The Intravenous Injection of One Massive Dose of Neo-Arsphenamin into Rabbits*

Experiment	Dosage Mg. per Kilogram	Weight in Grams	Time when Blood Was Taken	Mg. per 100 C.c. of Blood		
				Nonprotein Nitrogen	Urea Nitrogen	Creatinin
14	300	2,170	Before injection	36	18	1.5
		2,000	4 hours after injection	56	26	1.8
15	200	2,450	Before injection	41	22	1.7
		2,270	2.5 hours after injection	48	26	—
		2,095	5 days after injection	114	79	3.0
		1,825	8 days after injection	164	117	2.8
16	300	2,095	Before injection	36	13	1.9
		1,775	2.5 hours after injection	50	33	1.9
		1,745	4 days after injection	76	53	1.5
		1,670	7 days after injection	68	34	1.6
		1,740	10 days after injection	55	31	1.4
		1,840	13 days after injection	50	26	1.5
		1,880	17 days after injection	40	20	1.8
		1,920	20 days after injection	40	19	1.4

nonprotein nitrogen, urea nitrogen and creatinin content of the blood had greatly increased, the animal dying on the fourteenth day after injection. A necroscopic examination of the animal's kidneys showed parenchymatous nephritis.

Here we see that a dose representing two thirds of the maximum tolerated dose for rats produced nephritis and death in a rabbit. Animal 16, however, received three quarters of the maximum tolerated dose for white rats, and not only remained alive, but became almost normal with respect to the renal function within twenty days after injection and was returned to the stock. To explain this phenomenon we must assume an exceptionally strong vitality on the part of Rabbit 16, or a low renotropic effect of this particular lot of neo-arsphenamin.

The next experimental group consisted of animals injected with one large dose of neo-arsphenamin.

A perusal of Table 7 will disclose the fact already observed in our experiments with arsphenamin that about one half of the maximum

tolerated dose produces a slight temporary nephritis, which usually passes away within seven or eight days, although in one case it did not disappear until more than thirteen days after the injection. The case of Rabbit 20, in which 75 mg. caused a disturbance in the renal function lasting for eighteen days, may be explained by the pregnancy of the animal.

Our next step was to find out what effect, if any, multiple therapeutic doses had on the function of the kidneys in rabbits. As the largest single dose of neo-arsphenamin employed in human syphilotherapy is 0.9 gm., and as the weight of the average man is assumed to be approximately 60 kg., the average therapeutic dose would be 15 mg. per kilogram of body weight.

TABLE 7.—*The Intravenous Injection of One Large Dose of Neo-Arsphenamin into Rabbits*

Experiment	Dosage Mg. per Kilogram	Weight in Grams	Time when Blood Was Taken	Mg. per 100 C.c. of Blood		
				Nonprotein Nitrogen	Urea Nitrogen	Creatinin
17	150	2,390	Before injection	42	20	1.7
		2,050	2.5 hours after injection	52	27	1.8
		2,180	3 days after injection	53	25	1.6
		2,360	8 days after injection	27	13	1.7
18	150	3,245	Before injection	36	18	—
		3,175	2 hours after injection	40	23	—
		3,085	3 days after injection	47	26	1.8
		2,870	6 days after injection	55	31	1.8
		2,915	10 days after injection	44	23	—
		2,975	13 days after injection	39	20	1.7
19	150	3,085	Before injection	46	23	1.8
		3,040	2 hours after injection	50	25	2.1
		2,960	3 days after injection	56	29	2.0
		3,040	7 days after injection	42	20	1.9
20	75	3,740	Before injection	38	13	1.9
		3,535	2.5 hours after injection	41	17	1.7
		3,430	5 days after injection	55	34	1.6
		3,430	10 days after injection	46	25	1.5
		2,650*	13 days after injection	55	33	1.7
		2,830	17 days after injection	38	18	2.0

* The animal gave birth to young ones during the period intervening between this and the preceding injection.

The results indicated in Table 8 were to be expected. As in arsphenamin, a single therapeutic dose of neo-arsphenamin should produce little or no disturbance in the renal function of the animal. It was therefore rather surprising to notice a small rise in the nonprotein nitrogen content, etc., of the blood of Rabbit 21 a few hours after injection. But as the amounts of nonprotein and urea nitrogen in the blood of this particular animal were rather high even before the injection, we may ascribe the rise after injection to the weak or slightly diseased condition of the rabbit's kidneys before treatment, for the other two rabbits, each receiving twelve injections, remained normal in all respects.

We next turned to the mercurials, whose destructive action on the kidneys has been written about at length within the past few years. For this purpose we selected three organic mercury compounds: mercuric salicylate, and two new organic mercury compounds (sodium-oxymercury-orthonitrophenolate and the new mercurial recently elaborated in our laboratories 4-nitro-3, 5-bisacetoxymercuri-2-cresol).

The foregoing results clearly show the highly destructive effect of the mercurials on the kidneys, especially when injected intravenously.

TABLE 8.—*The Intravenous Injection of Multiple Therapeutic Doses of Neo-Arsphenamin into Rabbits*

Experiment	Dosage Mg. per Kilogram	Weight in Grams	Time when Blood Was Taken	Mg. per 100 C.c. of Blood		
				Nonprotein Nitrogen	Urea Nitrogen	Creatinin
21	15	2,500	Before injection	43	27	1.7
		2,415	2.5 hours after injection	50	30	2.3
		2,425	3 days after injection	38	20	1.7
			9 days after injection	—	—	—
		2,360	3.5 hours after second injection	42	26	1.8
		2,470	5 days after second injection	39	17	1.8
			8 days after second injection	—	—	—
		2,320	2.5 hours after third injection	48	26	2.0
		2,375	4 days after third injection	44	23	—
22	15*	2,505	Before injection	34	16	1.3
		2,515	2.5 hours after first injection	34	15	—
		2,500	4 days after first injection	30	15	1.3
		2,475	1 day after second injection	30	16	1.5
		2,490	1 day after third injection	38	18	—
		2,500	1 day after fourth injection	30	15	—
		2,470	1 day after fifth injection	29	14	—
		2,580	1 day after sixth injection	36	16	1.4
		2,505	1 day after seventh injection	32	15	1.4
		2,585	1 day after eighth injection	35	17	—
		2,620	1 day after ninth injection	36	18	1.5
		2,470	1 day after tenth injection	38	18	—
		2,480	1 day after eleventh injection	36	17	1.4
		2,500	1 day after twelfth injection	35	17	1.4
23	15*	2,110	Before injection	38	18	1.7
		2,070	1 day after first injection	40	18	1.6
		2,100	4 days after first injection	40	20	1.6
		2,070	1 day after second injection	38	19	1.6
		2,000	1 day after third injection	34	13	—
		2,010	1 day after fourth injection	38	18	—
		1,960	1 day after fifth injection	40	19	—
		2,115	1 day after sixth injection	35	16	1.5
		2,100	1 day after seventh injection	37	17	1.5
		2,100	1 day after eighth injection	37	18	—
		2,145	1 day after ninth injection	36	18	1.5
		2,010	1 day after tenth injection	35	16	—
		2,100	1 day after eleventh injection	37	18	1.6
		2,125	1 day after twelfth injection	38	19	1.6

* Injections given twice a week, three and four days apart.

Even as small a dose of mercuric salicylate as 3 mg. per kilogram trebled the nonprotein nitrogen content of the blood within twenty-four hours, and in the case of sodium-oxymercury-orthronitrophenolate all the three waste products increased almost tenfold, causing death on the third day. Rabbit 28 ate nothing after injection and passed little urine or feces, which accounts for his rapid loss of weight. Necroscopic examination showed acute tubular and glomerular nephritis with desquamation and early calcification.

Intramuscular injections, however, are less dangerous, a dose of 15 mg. per kilogram of mercuric salicylate causing less injury to the kidneys than 3 mg. per kilogram administered intravenously. Similarly, 1 mg. per kilogram of 4-nitro-3, 5-bisacetoxymercuri-2-cresol which proved to be an effective dose in the treatment of rabbit syphilis [11] has no observable destructive effect on the kidneys.

Our last series of experiments concerned itself with the organic compounds of bismuth which have recently been introduced into the treatment of syphilis. Of these two preparations have been tested, the sodium and potassium tartrobismuthate manufactured in France, and the potassium bismuth tartrate produced at our laboratories. The results are given in Table 10.

TABLE 9.—*Injection of Organic Mercury Compounds into Rabbits*

Substance	No. of Experiment	Dosage, Mg. per Kilogram	Route*	Weight in Gm.	Time when Blood Was Taken	Mg. per 100 C.c. of Blood: Nonprotein Nitrogen	Urea Nitrogen	Creatinin
Mercuric salicylate	24	3	I. V.	2,170	Before injection.....	29	14	—
				—	1 day after injection..	89	36	—
				—	4 days after injection.	50	22	—
				—	7 days after injection.	51	23	—
				2,250	9 days after injection.	32	13	—
	25	15	I. M.	2,280	Before injection.....	34	16	—
				—	1 day after injection..	45	19	—
				—	4 days after injection.	71	51	—
				2,360	7 days after injection.	38	16	—
4-nitro-3, 5 bisacetoxymercuri-2-cresol	26	1	I. M.	—	Before injection.....	38	14	
				—	1 day after injection..	36	15	
	27	1	I. M.	—	Before injection.....	38	16	
				—	1 day after injection..	36	15	
				—	7 days after injection.	34	16	
Sodium-oxymercury-orthonitrophenolate	28	5	I. V.	2,740	Before injection.....	26	11	1.4
				2,688	2 hours after injection	34	14	1.8
				2,315	1 day after injection..	161	118	3.9
				2,050	3 days after injection†	253	193	12.0

* I. V. indicates intravenous, and I. M., intramuscular.
† Death due to drug.

Either compound is highly toxic when administered intravenously, the maximum tolerated dose for the French product being about 5 mg. per kilogram and that of potassium bismuth tartrate, from 10 to 12 mg. It is therefore interesting to note that the rapid rise in the nonprotein nitrogen and urea content of the blood of Rabbits 29 and 30, which reached its peak on the third day, subsided quickly.

As is now well known, the intramuscular route is the one recommended for injecting organic bismuth compounds. In the case of the sodium-potassium tartrobismuthate, 40 mg. per kilogram produced a considerable but not dangerous rise of the nonprotein nitrogen and urea content of the blood, while a dose of 50 mg. per kilogram of potassium bismuth tartrate apparently had no deleterious effect on the kidneys. A

11. Raiziss and Severac: J. Lab. & Clin. Med. **9**:71, 1923.

dose of 100 mg. per kilogram, however, almost proved fatal, as it took a month for the kidneys to regain their normal function.

In this connection the delayed effect of the bismuth compound when injected intramuscularly is well worth observing. It is probably due to the slow absorption of the drug by the tissues. The latter should always be kept in mind when this route is chosen, and the recipient of the drug watched more carefully.

TABLE 10.—*Injection of Organic Bismuth Compounds into Rabbits*

Substance	No. of Experiment	Dosage, Mg. per Kilogram	Route	Weight in Gm.	Time when Blood Was Taken	Mg. per 100 C.c. of Blood: Non-protein Nitrogen	Urea Nitrogen	Creatinin
Sodium and potassium tartrobismuthate (French product)	29	3	I. V.	3,400	Before injection.....	34	14	1.5
				3,490	2.5 hours after injection	38	15	—
				3,250	3 days after injection...	76	51	2.0
				3,360	7 days after injection...	57	30	—
				3,300	10 days after injection...	50	28	1.8
				3,245	15 days after injection...	40	24	—
				3,290	18 days after injection...	40	21	—
				3,290	21 days after injection...	36	16	1.6
	30	4	I. V.	2,750	Before injection.....	38	16	1.4
				2,690	3.5 hours after injection	40	18	—
				2,500	3 days after injection...	132	74	1.9
				2,570	7 days after injection...	58	35	2.0
				2,700	10 days after injection...	50	30	1.7
				2,690	15 days after injection...	50	28	—
				2,630	18 days after injection...	38	15	1.3
	31	40	I. M.	2,630	Before injection.....	30	14	1.4
				2,620	2.5 hours after injection	32	15	1.4
				2,540	3 days after injection...	42	24	—
				2,545	7 days after injection...	29	13	1.4
Potassium bismuth tartrate (Dermatological Research Laboratories) oil suspension	32	3	I. V.	.....	Before injection.....	40	18	—
					1 day after injection....	51	—	—
					5 days after injection...	50	29	—
					6 days after injection...	48	22	—
					7 days after injection...	44	20	—
	33	100	I. M.	2,100	Before injection.....	40	16	—
				2,030	4 days after injection...	40	—	—
				2,020	6 days after injection...	42	19	—
				—	10 days after injection...	48	—	—
				—	13 days after injection...	50	—	—
				—	17 days after injection...	100	75	—
				1,440	28 days after injection...	127	94	—
				—	54 days after injection...	36	14	—
	34	50	I. M.	2,070	Before injection.....	36	14	—
				1,900	2 days after injection...	32	13	—
				1,900	4 days after injection...	32	12	—
				1,870	6 days after injection...	32	14	—
				—	11 days after injection...	36	16	—
				—	22 days after injection...	35	14	—

SUMMARY

1. Single massive doses of arsphenamin, approaching the maximum tolerated dose of the drug for white rats, in rabbits produce severe nephritis followed by death.

2. Single large doses of arsphenamin (about one half of the maximum tolerated dose) cause acute nephritis, which generally clears up within from ten to sixteen days.

3. Repeated, normal therapeutic doses of the drug do not disturb the renal function at all.

4. Large doses of o-aminophenol do not affect the kidneys, but produce severe reactions resembling those that frequently follow the admintration of arsphenamin.

5. Single massive doses of neo-arsphenamin (three times as large as that of arsphenamin) usually cause severe nephritis and death in rabbits, although some animals recover.

6. Single large doses of neo-arsphenamin (about ten times the normal therapeutic dose) produce acute nephritis, which clears up in a few days.

7. Repeated, normal, therapeutic doses of neo-arsphenamin do not injure the kidneys.

8. Whether compared weight for weight or on the basis of arsenic content, neo-arsphenamin is more than twice less injurious to the kidneys than arsphenamin.

9. Small doses of the organic mercurials tested proved greatly harmful to the kidneys, especially when injected intravenously, mercury compounds thus proving to be much more injurious than those of arsenic.

10. Both sodium-potassium tartrobismuthate (French product) and potassium bismuth tartrate (American) cause renal disturbances in animals when injected intravenously even when small doses are given. Intramuscularly, however, much larger doses may be given, with little or no injurious effect on the kidneys, although the latter may be slow in making its appearance and must be guarded against.

It is possible that many of the reactions following the administration of arsphenamin, neo-arsphenamin, etc., are only secondary manifestations resulting from injuries sustained by the kidneys. Great attention must therefore be paid to the renal function both before the injections are begun and during the course of treatment.

12. Of the three groups of compounds investigated, the mercurials are the most and the arsenicals the least injurious to the kidneys, while neo-arsphenamin is the least injurious. The latter can be given intravenously in relatively large quantities with the least amount of renal disturbance.

METABOLISM EXPERIMENTS IN INFANTILE ECZEMA *

PRELIMINARY REPORT

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NEW YORK

It had been the custom in the past, at the New York Skin and Cancer Hospital, where literally hundreds of cases of infantile eczema of every sort and description are seen yearly, to put these infants on a modified and attenuated diet, consisting of low fat and sugar content and a lessened quantity. In addition to this, local applications, lotions, ointments, etc., were employed. This treatment is a recognized one for infantile eczema throughout the country. In the case of breast nursing infants, the amount and quality of the food was reduced by lengthening the intervals between nursings, and by reducing the time of nursing, or by nursing on both breasts at the same feeding. In the case of bottle fed infants, it was our practice first to reduce the fat in the milk, and then if no improvement resulted, to reduce the sugar. In other words, one perhaps could cure the eczema, but *might* kill the child. In this study, we were chiefly interested in infants of 1 year or under, so did not go into the mixed diets of older children.

In the attempt to throw some light on this puzzling condition, we have been running two parallel experiments: first, the metabolism of the stools and urine in patients with eczema, who were fed on high fat or sugar (with proper regard to the external eczematous condition); and second, clinical observations on dispensary patients with eczema over a period of two years, all of them fed on diets proper for their weights and ages, without any reduction of the fat or sugar, and treated as uniformly as possible with external applications. Our results ran quite parallel. In other words, we could see no definite change in the eczema by the addition or subtraction of fat or sugar within certain

* From the Research Laboratory of the New York Skin and Cancer Hospital.

limits, and the eczema cleared up just as readily as in those cases in which these elements had been withdrawn. In the preliminary study we performed no protein sensitization tests, being interested primarily in the fats and sugars.

PART I

This investigation consists of a study of the protein, fat, carbohydrate and mineral metabolism in babies with eczema. The results with one infant who had no skin disease are almost similar to the results obtained with the other infants.

METHODS

The metabolism bed was similar to the beds used at the Babies' Hospital, New York, and described by Courtney and Fales.[1] To enable the accurate and separate collection of urine and feces, male infants were studied. The twenty-four hour specimen of urine was collected every day, the separate specimens being placed in a bottle in the icebox as soon after voiding as possible.

The following determinations were made on each twenty-four hour specimen: total nitrogen, ammonia, urea, uric acid, creatinin, total sulphates and sugar. The methods, except that for sugar, are described in the St. Luke manual; that for sugar is in Myer's "Chemical Analysis of Blood and Urine." The results tabulated are:

1. Quantity and specific gravity.
2. The weights of ammonia, urea, uric acid, creatinin and total nitrogen excreted in twenty-four hours.
3. The nitrogen partition, that is, the percentage of total nitrogen, which is ammonia nitrogen, urea nitrogen, uric acid nitrogen and creatinin nitrogen.
4. Weight of sugar in terms of glucose in the twenty-four hour specimen.
5. Weight of sulphur in total sulphates.

The feces were collected in a diaper and transferred to a wide-mouthed bottle and placed in the icebox. The twenty-four hour specimen was then placed in an evaporating dish and dried on a water bath. When it was thoroughly dried, it was powdered, put into a weighing bottle and set aside. Before analysis, the sample was transferred to an evaporating dish, dried in the oven at 98 to 100 C. and weighed.

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1. Courtney, A. M., and Fales, H. L.: A Comfortable Arrangement for the Separate Collection of Urine and Feces of Male Infants, Am. J. Child. Dis. **10**:410 (Dec.) 1915.

The following determinations were made on each twenty-four hour specimen of dried stools: total nitrogen, ash, total fat,[2] total carbohydrate (all carbohydrates were hydrolyzed to glucose with hydrochloric acid and the glucose determined by titration with Benedict's solution), and the calcium in the ash (McCrudden's method, described by Hawk[3]). The results tabulated are:

1. The weight of total nitrogen, total fat, ash and calcium oxid in the twenty-four hour specimen.
2. Percentage of dried weight of feces of these constituents.
3. The value of the total carbohydrate is expressed in terms of glucose.

DIETS

P. S.: Nov. 9, 1921: Formula, 32 ounces (⅔ milk, ⅓ granum water)
2 tablespoonfuls potato
1 tablespoonful spinach or string beans
6 tablespoonfuls farina
Nov. 11, 1921: Add 1 tablespoonful cane sugar
Nov. 13, 1921: Add 2 tablespoonfuls cane sugar

W. K.: Nov. 20, 1921: Formula, 35 ounces (25 ounces skimmed milk, 10 ounces granum water); 9 a. m. and 6 p. m., 1 tablespoonful cream of wheat
Nov. 24, 1921: Add 1 tablespoonful cane sugar
Nov. 27, 1921: Add 2 tablespoonfuls cane sugar

M. T.: Dec. 27, 1921: Formula, 32 ounces (24 ounces 1 per cent. milk, 8 ounces water, 2 tablespoonfuls of wheat flour)
4 ounces orange juice
6 tablespoonfuls farina
2 tablespoonfuls spinach
1 slice zweibach
Dec. 30, 1921; Jan. 3, 1922; Jan. 6. Jan. 10, Jan. 14, Jan. 18, 1922: Add ⅓ ounce cane sugar, making 2 ounces sugar

H. C.: Jan. 27, 1922: Formula, 35 oz. (24 oz. milk, 11 oz. granum water, 2 tablespoonfuls milk sugar)
Jan. 30, Feb. 3 and Feb. 8: Add ⅓ oz. milk sugar, making 1⅔ oz. sugar

DISCUSSION OF RESULTS (SEE TABLES 9 AND 10)

The results show an increase in ammonia with the addition of sugar and with the length of time the specimens were collected in the case of P. S. and W. K. In the case of M. T., the ammonia increases and decreases irregularly with the addition of sugar. There is no relation in any case between the increase of ammonia and the addition of sugar. The normal value given for ammonia nitrogen is from 4 to 6 per cent.

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2. Holt, Courtney and Fales: Fat in Dried Feces, Am. J. Dis. Child. **17:** 38 (Jan.) 1919.

3. McCrudden, in Hawk: Physiological Chemistry, Philadelphia, P. Blakiston's Son & Co., 1917.

TABLE 1.—*Analysis of Twenty-Four Hour Specimens of Urine of S. H., Aged 5-6 Months*

Date 1922	Quantity, C c.	Specific Gravity	Ammonia, Gm	Urea, Gm.	Uric Acid, Gm.	Creatinin, Gm.	Total Nitrogen, Gm.	Ammonia Nitrogen, Percentage Total	Urea Nitrogen, Percentage Total	Uric Acid Nitrogen, Percentage Total	Creatinin Nitrogen, Percentage Total	Sugar Glucose	Total Sulphate
Dec. 6–7	484	1.008	0.329	2.039	0.078	0.020	1.193	21.4	76.0	2.18	0.62	0.185	....
Dec. 7–8	555	1.007	0.520	1.794	0.161	0.040	1.561	27.5	53.0	3.43	0.95	0.111	....
Dec. 8–9*													
Dec. 9–10	359	1.007	0.216	1.844	0.077	0.029	1.507	25.6	56.4	1.64	0.71	0.116	....
Dec. 10–11	460	1.006	0.259	2.015	0.125	0.021	1.301	16.4	71.9	3.19	0.59	0.111	....
Dec. 11–12	432	1.006	0.393	1.896	Very low	0.035	1.307	24.0	67.4	Very low	0.90	0.065	0.115
Dec. 12–13	580	1.008	0.313	1.761	Very low	0.034	1.399	22.3	58.1	Very low	0.90	0.046	0.261
Dec. 13–14	484	1.006	0.252	1.516	Very low	0.017	0.989	25.4	71.6	Very low	0.63	0.036	0.184
Dec. 14–15	474	1.008	0.346	2.051	0.059	0.029	1.507	22.8	63.5	1.30	0.46	0.091	0.130
Dec. 15–28*													
Dec. 27													
Dec. 28–29	528	1.012	0.416	2.609	0.072	0.023	1.611	21.2	61.1	1.48	0.53	0.064	0.067
Dec. 29–30	464	1.006	0.398	1.060	0.052	0.010	1.065	30.7	46.8	1.52	0.37	0.145	0.077
Dec. 30 to Jan. 2, 1923*													
Jan. 2–3	342	1.011	0.683	1.352	0.147	0.018	1.335	42.1	47.3	3.66	0.50	0.104	0.077
Jan. 3–4	330	1.010	1.314	0.892	0.071	0.011	0.646	23.0	63.8	4.01	0.63	0.083	0.036
Jan. 4–5	788	1.008	0.782	1.938	0.148	0.026	1.765	46.6	50.4	4.06	0.71	0.167	0.149
Jan. 5–6	350	1.010	0.443	0.576	0.081	0.011	0.882	21.4	30.6	3.05	0 45	0.053	0.038
Jan. 6–7*													
Jan. 7–8	466	1.011	0.792	2.129	0.082	0.019	1.614	24.7	61.5	2.32	0.60	.....	0.151
Jan. 8–9	530	1.011	1.203	4.318	0.122	0.021	3.001	33.02	65.2	2.28	0.43	0.214	0.252
Jan. 9–10	470	1.010	1.118	3.070	0.101	0.015	2.593	28.7	57.0	1.31	0 22	0.451	0.140
Jan. 10–12													
Jan. 11													
Jan. 12–13	370	1.008	0.819	1.428	0.065	0.017	1.269	38.2	53.03	1.71	0.50	0.237	0.151
Jan. 13–14	640	1.009	1.014	2.012	0.177	0.017	2.168	33.8	45.1	2.62	0.29	0.129	0.144
Jan. 14–15	450	1.010	1.027	2.088	0.084	0.012	1.958	23.04	49.9	1.43	0.22	0.123	0.149
Jan. 15–16	480	1.007	0.722	0.538	0.103	0.015	0.984	63.4	25.1	3.48	0.56	0.144	0.055
Jan. 16–25*													
Jan. 25–26	246	1.016	0.586	2.24	0.077	Very low	1.620	30.0	46.7	1.58	Very low	0.125	0.137
Jan. 26–28*													
Jan. 28–29	166	1.007	0.312	0.542	0.045	0.0084	0.721	35.5	35.1	2.07	0.36	0.038	0.031
Jan. 29–30	255	1.006	0.487	0.331	0.054	0.010	1.407	28.5	11.0	1.27	0.26	0 046	0.08
Jan. 30–31	384	1.006	0.492	0.132	0.057	0.031	0.774	52.3	8.0	2.45	0.15	0.127	0.042
Jan. 31 to Feb. 1	228	1.006	0.324	0.122	0.034	0.013	0.501	53.1	11.9	2.26	0.09	.....	0.036
Feb. 1–2	214	1.008	0.395	0.137	0.029	Very low	0.456	?	14.1	2.12	Very low	0.142	0.031

* No specimens collected.

TABLE 2.—*Analysis of Twenty-Four Hour Specimens of Stools—Baby Samuel Holzman, aged 5-6 Months*

Date, 1922	Dried, Gm.	Nitrogen, Gm.	Nitrogen, Percentage	Ash, Gm.	Ash, Percentage	Calcium Monoxid, Gm.	Calcium Monoxid, Percentage	Calcium Monoxid, Percentage of Ash	Fat, Gm.	Fat, Percentage	Carbohydrate, Gm. (Glucose)
Dec. 6–7	7.705	0.173	2.23	1.849	21.1	1.15	15.0	62.5	1.772	22.9	.....
Dec. 7–8	3.469	0.109	3.10	0.971	28.0	0.642	18.5	66.0	0.676	19.5	.....
Dec. 8–9*											
Dec. 9–10	5.845	.....	....	1.929	33.1	0.964	16.5	49.4	1.198	20.5	0.309
Dec. 10–11†	5.200	.....	....	?	?	0.936	18.0	....	1.950	37.5	0.374
Dec. 11–12	4.845	.....	....	1.308	27.0	0.485	10.0	37.1	1.76	27.6	.....
Dec. 12–13	7.230	0.272	3.76	2.024	28.0	1.033	14.3	46.1	2.060	28.5	0.311
Dec. 13–14	3.185	.....	....	.....	....	...	....	....	0.987	30.0	.....
Dec. 14–15	4.438	.....	....	0.932	21.0	0.615	13.8	65.9	0.621	13.9	0.249
Dec. 15–28											
Dec. 27†											
Dec. 29–30	0.850	.....	....	.....	....	.....	....	....	0.170	21.2	.....
1923											
Jan. 2–3	9.110	0.307	3.26	2.241	24.6	1.406	15.4	62.6	2.049	22.3	0.261
Jan. 3–4	2.990	.....	....	.....	....	.....	....	....	0.762	25.5	.....
Jan. 4–5‡	5.165	0.197	3.81	2.298	44.5	1.384	26.8	62.3	1.342	26.0	0.243
Jan. 5–6	5.590	0.165	2.95	0.950	17.0	0.692	12.4	72.8	.....	....	0.390
Jan. 6–8*											
Jan. 8–9	6.550	0.229	3.34	0.937	14.3	?	?	?	2.652	40.5	0.381
Jan. 9–10	4.105	.....	....	.....	....	.....	....	....	1.806	44.0	0.197
Jan. 10–12*											
Jan. 11‡											
Jan. 12–13	10.950	0.231	3.02	1.565	14.3	1.117	10.2	71.3	4.445	40.6	0.548
Jan. 13–14	9.210	.....	....	1.289	14.0	0.958	10.4	74.3	4.651	50.5	0.480
Jan. 14–15	7.884	.....	....	1.947	?	1.049	?	53.3	4.525	57.4	0.312
Jan. 15–16	6.572	0.219	3.33	0.914	13.9	0.551	8.4	60.3	4.074	62.0	0.281
Jan. 25–26‡	5.916	0.189	3.19	0.852	14.4	0.372	6.3	42.5	2.543	43.0	0.140
Jan. 29–30	5.883	0.159	2.72	0.312	?	?	?	?	4.206	71.5	0.280
Jan. 30–31	5.306	.....	....	0.743	14.0	0.371	7.0	50.0	2.918	55 0	0.289
Jan. 31–Feb. 1	4.143	.....	....	0.626	15.1	0.394	9.5	63.0	2.485	60.0	0.213

* No specimens collected.
† Sugar increased in diet.
‡ Fat increased in diet.

of the total nitrogen. It is of interest to notice that the first specimens collected gave normal values for ammonia nitrogen; for instance, the ammonia nitrogen in the case of P. S. (first specimen) is 5.5 per cent. of total nitrogen, of W. K., 7.4 per cent.; of M. T., 6.8 per cent. In the case of W. K., the ammonia nitrogen increased on the second day to 17.6 per cent. of total nitrogen; on the third day it was 14.1 per cent., and on the fourth day 14.6 per cent., while no sugar had been added to the formula. In the case of M. T., the ammonia nitrogen was 14.5 per cent. of the total nitrogen on the second day and 14.8 per cent. on the third day without the addition of sugar to the diet.

The question arose: Does the constrained position and lack of exercise of the infant affect its metabolism appreciably and does this cause an increase in ammonia? This question could be settled by collecting specimens for one day, then allowing free movements for several days, and again collecting a twenty-four hour specimen.

It was also noticed that the carbohydrate in the stools varied and that the variation was in the same direction as in the case of the ammonia; that is, usually when the ammonia nitrogen in the urine increased there was also an increase in the carbohydrate content of the feces.

The urea nitrogen, uric acid and creatinin content of the urine are approximately normal except when the ammonia content is high, in which case the urea nitrogen content is low. The normal values are: urea nitrogen, from 78 to 88 per cent. of the total nitrogen; uric acid nitrogen, 1.3 per cent. of total; creatinin nitrogen, 1.6 per cent of total. These values are average values.

In the case of H. C., who was 6 months old, weighed 11 pounds (5 kg.), and had no skin disease, the results are similar to the others. The ammonia nitrogen is much higher than normal and varies irregularly with the addition of milk sugar.

In all cases, the urine specimens were neutral or alkaline and contained heavy deposits of amorphous and crystalline phosphates.

FECES

Holt, Courtney and Fales give the following average values for stools of normal infants:

Diet	Fat Per Cent. Dried Weight	Nitrogen Per Cent. Dried Weight	Ash Per Cent. Dried Weight
Modification of cow's milk.....	36.2	4.1	24.1
Milk plus cereal.....	30.7	..	...
Mixed diet	18.0	..	...

The values obtained in this investigation are similar.

	Fat Per Cent. Dried Weight	Nitrogen Per Cent. Dried Weight	Ash
P. S.	18.3	3.9	23
M. T.—mixed diet	19.0	4.4	16
W. K.—milk cereal	29.8	4.9	24

The results which would serve as a more satisfactory basis for comparison with normal values are the values for the percentage of protein, fat, carbohydrate and ash retained by the infants. To calculate these values it is necessary either to analyze a sample of the diet or to calculate the composition of the diet from tables. Since in this work, the constituents of the diet were not weighed, it was not possible to calculate the protein, fat, carbohydrate and ash intake.

Although the output of ammonia in urine was abnormal, similar results were obtained with infants with and without eczema. Courtney [7] gives a high value for ammonia nitrogen in a case of malnutrition.

The other substances determined agree approximately with normal values found in the literature.

No values for the carbohydrate output in the feces were found, so that these results could not be compared with normal values.

Part II—Study of S. H.

The present problem is a continued investigation of the protein, fat, carbohydrate and mineral metabolism in cases of infantile eczema, begun in November, 1921, by Miss Johann. The case under consideration is that of S. H., aged 5 months, weighing 13 pounds (6 kg.) and admitted to the hospital on Nov. 24, 1922. The rash was principally on the face and head. With the exception of a lotion of calamin and zinc applied to the face and head and Lassar's paste to the buttocks, no other medication was given. The investigation was undertaken to study what influence the increase of sugar and fat in the diet, especially the latter, had on the child's metabolism, and whether or not that had any effect on the disappearance or continuance of the eczematous symptoms. The work mentioned above had been done with merely an increase in sugar, and the results showed in two out of three cases an increase in ammonia, but no direct relationship was noticed. In the present case, abnormal amounts of ammonia nitrogen were excreted, but since 1 ounce of sugar was added at the very beginning, no comparison can be made. However, as in the previous work, it was noted that there was no direct relationship between the amount of sugar added and the increase in ammonia excretion.

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7. Courtney, Angelia M.: Studies on Infant Nutrition, Am. J. Dis. Child. **1**:321 (May) 1911.

The formulas were as follows:

S. H. Dec. 4, 1922: Milk.....18 ounces
Barley water ..22 ounces
Cane sugar ... 1 ounce
Cream of wheat. 4 teaspoonfuls (80 gm.)
Orange juice...½ ounce

Dec. 10: Sugar increased, ⅓ ounce

Dec. 27: Sugar increase, ⅓ ounce

Dec. 29: Sugar increase, ⅓ ounce

Jan. 4, 1923: Fat increased by:
Top half of milk..... 16 ounces
Barley water ..... 24 ounces
Cane sugar ..... 2 ounces
Cream of wheat..... 2 teaspoonfuls
Orange juice ..... 1 ounce

Jan. 11, 1923: Fat increased by:
Top half milk..... 18 ounces
Barley water ..... 22 ounces
Other items the same

Jan. 25, 1923: Fat increased by:
Top half milk..... 20 ounces
Barley water ..... 20 ounces
Other items the same

RULES FOR METABOLIC WORK

For proper cooperation with the chemist, the following rules must be carried out by nurses, *without fail:*

1. Careful watch must be made frequently, especially by night nurse to see that rubber connection is tight; if loose at any time, this *must be reported.*
2. The 24-hour specimens are collected from 9 a. m. exactly to 9 a. m. daily, unless otherwise informed. Please always use the same timepiece.
3. Urine must be emptied in labeled bottle in icebox at 5 p. m. and 9 a. m.
4. Stools to be placed in casserole at the same time.
5. Formulas to be carefully measured, and *amount taken at each feeding recorded and reported on slips provided by chemist.* It is especially important that this last procedure be carried out because on it depends the calculation of the intake and retention.

Daily slips to be filled out by nurses in metabolic cases

Name .....
Date.....Weight at 9 a. m.
Condition .....
(Anything unusual or improvement)

Amounts taken at feedings

Time	*Amounts*	*Nurse*
Any Change in Formula		

9 a. m.
.....(Nurse)

METHODS

The metabolism bed was similar to those used at the Babies' Hospital, described by Courtney and Fales.[1] The twenty-four hour specimens of urine were collected and placed in the icebox as soon after voiding as possible, and all determinations were completed the same day on substances which would deteriorate. Extreme care was taken to obtain accurate specimens, and doubtful specimens were discarded. Lack of nurses was the reason in most cases for not collecting the specimens.

The following determinations were made on each twenty-four hour specimen: total nitrogen, ammonia, urea, uric acid, creatinin, total sulphates and sugar. The methods, except that for sugar determination, are described in the St. Luke's Hospital Manual;[4] for sugar, in Myers' "Chemical Analysis of Blood."[5] The results tabulated are:

1. Quantity and specific gravity
2. Weight in grams of ammonia, urea, uric acid, creatinin and total nitrogen excreted in twenty-four hours
3. The nitrogen partition, that is, percentage of total nitrogen which is creatinin nitrogen, ammonia nitrogen, urea nitrogen, and uric acid nitrogen
4. Weight of sugar in terms of glucose
5. Weight of sulphur in total sulphates

The feces were collected in a diaper, transferred to a casserole, and directly dried on a water bath, powdered and stored in a stoppered bottle until ready to be analyzed. Before analysis the sample was dried in the oven at from 98 to 100 C. for two hours, and then weighed. The determinations made were: total nitrogen, ash, fat,[2] total carbohydrates (hydrolyzed to glucose with HCl and determined by titration with Benedict's solution), total ash, and the calcium in the ash (McCrudden's method[3]).

The results tabulated are:

1. The weights in grams of total nitrogen, fat, ash and calcium oxid
2. Percentage of the dried weights of feces of these constituents
3. The value of the total carbohydrate expressed in terms of glucose.

DISCUSSION OF RESULTS IN S. H.

The results, as in the previous investigation, show an abnormal excretion of nitrogen in the form of ammonia, but since sugar was added at the very beginning of the experiment, no comparison can be

4. St. Luke's Hospital Manuel, Wood, Vogel and Famulener (Doughterty) Publisher, New York, 1922.

5. Myers, Victor C.: Practical Chemical Analysis of Blood, published by the C. V. Mosby Company.

made. However, as noted in the previous work, there was no direct relation between the amount of sugar added and the increase in ammonia.

As to the increase of fat, the figures show the following results: Before the increase, the average value for ammonia nitrogen excretion was 26.8 per cent. of the total nitrogen. The average normal value is 4.5 per cent. After the first increase, the value was 26.9 per cent.; the second increase, 31.2 per cent. and the third increase, 32 per cent. In estimating these averages all questionable figures were discarded. Therefore after the second increase in the fat intake there seems to be a slight increase in the amount of nitrogen excreted in the form of ammonia. However, the figures vary so from day to day that no comparison or relationship can be made with this one case. The average values for uric acid nitrogen are somewhat higher than the normal 1.3 per cent. and the creatinin nitrogen in every case is lower than the normal 1.6 per cent. Nevertheless, there seems to be no definite relationship between this excretion and the sugar and fat increase.

Before the fat was increased, the percentage of the dried weight of a stool was 24.6 per cent., after the fat was increased the average was 51.7 per cent., or 42.2 per cent., 50.7 per cent. and 62.2 per cent. when, respectively, 16 ounces, 18 ounces and 20 ounces of the top half of milk was used. Averaging figures on all diets, 44.9 per cent. is obtained, which is higher than the 36.1 per cent. given by Courtney and Fales.[2] However, since here there is only one case to be considered, it might be well to compare with individuals with similar fat intakes reported by them.

TABLE 3.—*Table from Article of Courtney and Fales, Am. J. Dis. Child., Vol. 17, 1919*

Fat Intake, Gm.	Fat, Percentage Dried Weight of Stool
22	25.7
30.5	51
Case of S. H. Case under consideration	
20.5	24.6
34.4	42.2
34.9	50.7
35.1	62.2
Average	44.9

Since the constituents of the diets were not weighed, the fat intake [6] could only be obtained approximately, yet in spite of this the similarity with other individual cases previously reported is close. Agreeing with them, there seems to be no definite relationship between the daily fat intake and the percentage of fat in the stool.

6. Calculated from values in "Laboratory Handbook for Dietetics" by Professor Mary Swartz Rose of Columbia University, New York, The MacMillan Company.

In regard to the percentage of retention of fat, the following results were obtained by Courtney and Fales:

TABLE 4.—*Table from Article of Courtney and Fales, Am. J. Dis. Child., Vol. 17, 1919*

Fat Intake		Fat in Stool, Gm. Daily	Fat Retention	
Gm. Daily	Daily Average		Gm. Daily	Percentage
14.6 - 36.5	26.7	2.33	24.4	91.3

TABLE 5.—*Case of S. H.*

Formulas Containing	Fat Intake, Gm. Daily	Fat in Stool, Gm. Daily	Fat Retention, Gm. Daily	Percentage Retention
16 oz. whole milk	20.5	1.38	19.2	93.5
16 oz. milk, half top	34.4	2.23	31.6	91.8
18 oz. milk, half top	34.9	4.42	30.4	86.2
20 oz. milk, half top	35.1	3.45	31.9	90.3
Average	31.2	2.87	28.3	90.5

Courtney and Fales give as a retention range from 85.8 to 97.4 per cent. By consulting Table 6, one can readily see that all results fall within these figures with the exception of two days, one being 85.2 per cent. and the other 83.8 per cent. Agreeing with them, there seems to be no striking relation between the fat intake and the percentage of fat retained. They found it to happen only when the intake was abnormally low.

TABLE 6.—*Fat Retention (Case of S. H.)*

Date, 1922	Fat Intake, Gm.	Fat Output, Gm. Daily	Fat Retention, Gm. Daily	Percentage Retention
Dec. 6- 7	20.536	1.772	18.764	91.3
Dec. 7- 8	20.536	0.676	19.760	96.2
Dec. 9-10	20.536	1.198	19.338	91.3
Dec. 10-11	20.536	1.950	18.586	90.5
Dec. 11-12	20.536	1.760	19.360	94.4
Dec. 12-13	20.536	2.060	18.476	89.9
Dec. 13-14	20.536	0.987	19.549	95.1
Dec. 14-15	20.536	0.621	19.915	96.4
1923				
Jan. 2- 3	20.536	2.049	18.487	90.4
Jan. 3- 4	20.536	0.762	19.774	96.2
Jan. 4- 5	20.536	1.342	18.194	88.6
Jan. 8- 9	34.442	2.652	31.790	92.3
Jan. 9-10	34.442	1.806	32.536	94.4
Jan. 12-13	34.916	4.445	30.471	87.3
Jan. 13-14	34.916	4.651	29.265	83.8
Jan. 14-15	34.916	4.525	30.391	86.7
Jan. 15-16	34.916	4.074	30.842	88.3
Jan. 25-26	35.192	2.543	32.649	92.2
Jan. 29-30	35.192	4.206	30.986	88.0
Jan. 30-31	35.192	2.918	32.174	91.0
Jan. 31-Feb. 1	35.192	2.485	32.707	92.1

The average percentage of nitrogen of dried weight of stool, 3.5 per cent., is similar to the figures given by Courtney and Fales for infants on a diet of modification of cow's milk. No figures for carbohydrate output were found so that these results could not be compared with normal values.

The average ash percentage of dried weight of stool as given by Courtney and Fales for infants on modification of cow's milk is 24.1 per cent.; the average in this investigation is 26.1 per cent.

TABLE 7.—*The Relation of Calcium Absorption to Calcium Intake Per Kilogram (Case of S. H.)*

Diet	Calcium Oxid Intake, Gm. Daily	Calcium Oxid Output, Gm. Daily	Calcium Oxid Retained, Gm. Daily	Calcium Oxid Retention, Percentage	Calcium Oxid Intake, Gm. per Kilogram	Calcium Oxid Retention, Gm. per Kilogram
18 oz milk.....	1.307	0.957	0.35	26.7	0.21	0.057
16-20 oz. half top milk.....	0.769	0.691	0.078	10.1	0.13	0.013
Average Courtney-Fales....	.....	0.70	.....	35-55	....	0.089

By comparing the estimates in Table 7 with normal figures, one can see that the percentage of calcium absorbed is lower than the average.

TABLE 8.—*Calcium Excretion in Relation to Calcium and Fat Intake*

Calcium Oxid Intake, Gm. Daily *	Fat Intake, Gm. Daily	Calcium Oxid Output, Gm. Daily	Fat Output, Gm. Daily
1.307	20.5	0.957	1.38
0.769	34.8	0.691	3.36

* Calculated from values in "Laboratory Manual for Dietetics" by Prof. Mary Swartz Rose, with suggestions by Prof. Henry C. Sherman of Columbia University.

Agreeing with Courtney and Fales, there is no constant relationship between the amount of fat intake and the calcium excreted; the higher the calcium intake the higher the excretion and vice versa.

CONCLUSIONS AND SUMMARY: CASE S. H.

In this case of infantile eczema, the following conclusions can be made regarding metabolism:

1. The amount of nitrogen excreted in the form of ammonia is abnormal, but there seems to be no definite relationship between this excretion and the sugar and fat increase; uric acid nitrogen is somewhat higher and creatinin nitrogen lower (Table 1).

2. The fat metabolism appears to be normal; the percentage retained, 90.5 per cent., compares very well with Courtney and Fales' figure of 91.3 per cent. (Tables 2, 3, 4, 5 and 6).

3. The average percentage of nitrogen in stool, 3.5, is similar to the figure given by Courtney and Fales, as is also the ash, 26.1 per cent. (Table 2).

4. The percentage of calcium absorbed is lower than the average of 0.089 gm. per kilogram and the excretion is higher but similar to the figure of 0.86 given by Courtney and Fales in the case of rachitic infants. The question arises: Has infantile eczema anything to do with calcium metabolism (Tables 2, 7, 8)?

5. Since the child began to recover and became practically cured after the increase in fat, one could not say that the fat in this case had any destructive effect. Whether it was something in the balance of the diet or the fact that the child did not irritate itself much that effected the cure, remains a question.

Part IIa. Study of E. D.

The case under consideration is that of E. D., aged 3 months, weight 11½ pounds (5 kg.), who was admitted to the hospital on April 18, 1923. The eruption extended over the face and scalp with yellow crusting; there was also moderate infiltration of the skin of the arms. Throughout the treatment a lotion of calamin and zinc was applied to the body, while the medication on the face was somewhat changed.

The diet before admittance to the hospital consisted of:

Whole milk	20 oz.
Water	14 oz.
Cane sugar	3 oz.

On admittance the formula was changed to:

Whole milk	20 oz.
Barley water	10 oz.
Milk sugar	2½ oz.

The diet was continued for eight days, and the child did not seem to improve, although he maintained his weight. On April 26, the fat in the diet was increased to 7 per cent. by using the top half of the milk.

Top half of milk	20 oz.
Barley water	10 oz.
Milk sugar	2½ oz.

A day later, although the child regurgitated a small amount of the feeding, his face began to clear up, and later the body; the whole skin condition tended toward improvement. After a period of seven days, on May 3, the formula was changed again:

Top half of milk	22 oz.
Barley water	8 oz.
Milk sugar	2½ oz.

The condition tended to improve for five days in spite of the change, and weight was not only maintained but increased. Then suddenly the condition became bad; the eruption was watery and red, and the child was put on the original diet with the smaller fat percentage of:

Milk	20 oz.
Barley water	10 oz.
Milk sugar	2½ oz.

Table 9.—Analysis of Twenty-Four Hour Specimen of Urine

TABLE 9.—*Analysis of Twenty-Four Hour Specimen of Urine*

	Date, 1921	Quantity, C.c.	Specific Gravity	Ammonia, Gm.	Urea, Gm.	Uric Acid, Gm.	Creatinin, Gm.	Total Nitrogen, Gm.	Ammonia Nitrogen, Percentage Total	Urea Nitrogen, Percentage Total	Uric Acid, Percentage Total	Creatinin Nitrogen, Percentage Total	Sugar, Gm.	Total Sulphate (Gm. Sulphur)
P. S., 1 year of age; urine	Nov. 9–10	516	1.014	0.144	3.46	0.119	0.056	2.16	5.5	75.0	1.83	0.96	.....	0.119
	Nov. 10–11	540	1.014	0.117	3.98	0.085	0.054	2.24	4.3	83.0	1.3	0.9	.....	0.115
	Nov. 11–12*	530	1.013	0.306	4.58	0.120	0.069	2.76	9.1	77.5	1.45	0.93	.....	0.161
	Nov. 12–13	480	1.012	0.343	3.40	0.084	0.040	2.40	11.75	66.3	1.17	0.61	.....	0.127
	Nov. 13–14*	900	1.011	0.355	5.56	0.141	0.076	3.51	8.32	74.1	1.34	0.80	.....	0.189
	Nov. 14–15	530	1.011	0.252	3.23	0.083	0.045	2.19	9.50	68.9	1.26	0.77	0.080	0.111
	Nov. 15–16	710	1.013	0.845	4.22	0.180	0.077	3.46	20.1	56.9	1.73	0.82	0 121	0.196
	Nov. 16–17	570	1.020	0.841	4.99	0.148	0.053	3.43	20.2	67.9	1.44	0.57	0.080	0.218
	Nov. 18–19	670	1.017	0.105	5.09	0.191	0.032	3.58	24.2	66.5	1.78	0.33	.....	0.202
W. K., 6 mos.	Nov. 20–21	370	1.014	0.141	2.59	0.120	0.0380	1.57	7.4	77.1	2.54	0.90	0.040	0.066
	Nov. 21–22	366	1.017	0.384	3.10	0.126	0.042	1.79	17.6	81.0	2.3	0.87	0.046	0.104
	Nov. 22–23	470	1.014	0.316	2.76	0.162	0.029	1.85	14.1	70.0	2.9	0.57	0.070	0.089
	Nov. 23–24	530	1.013	0.332	2.98	0.138	0.037	1.87	14.6	74.4	2.5	0.73	0.077	0.095
	Nov. 24–25*	500	1.012	0.374	2.46	0.109	0.037	1.62	19.0	70.9	2.2	0.85	0.060	0.085
	Nov. 25–26	390	1.017	0.600	2.72	0.111	0.019	1.97	25.1	64.5	1.88	0.36	0.029	0.105
	Nov. 26–27	440	1.017	0.524	2.48	0.117	0.037	1.95	22.1	59.3	2.0	0.71	0.049	0.106
	Nov. 27–28*	430	1.017	0.728	3.15	0.113	0.014	2.24	26.8	65.6	1.68	0.23	0.049	0.133
	Nov. 28–29	520	1.014	0.700	2.84	0.101	0.040	2.16	26.7	61.3	1.55	0.69	0.039	0.117
	Nov. 29–30	495	1.018	0.653	3.21	0.112	0.028	2.41	22.3	62.2	1.55	0.44	0.063	0.139
M. T., 14 mos.; (3 specimens discarded)	Dec. 27–28	410	1.020	0.200	3.68	0.171	0.023	2.43	6.79	70.9	2.34	0.35	0.049	0.127
	Dec. 28–29	510	1.013	0.309	2.17	0.035	0.063	1.76	14.5	57.7	0.67	1.32	0.064	0.070
	Dec. 29–30	200	1.021	0.182	1.36	0.062	0.022	1.014	14.8	62.4	2.02	0.80	0.028	0.045
	Dec. 30–31*	308	1.013	0.331	0.830	0.088	0.036	0.897	30.3	43.3	3.27	1.47	0.032	0.039
	Dec. 31–Jan. 1	210	1.016	0.457	0.315	0.072	0.005	0.706	53.2	20.8	3.4	0.24	0.017	0.035
	Jan. 1– 2	396	1.012	0.490	0.616	0.032	0.057	0.987	40.9	29.2	1.09	2.15	0.042	0.048
	Jan. 2– 3	636	1.010	0.320	1.068	0.014	0.058	1.122	23.5	44.5	0.41	1.91	0.065	0.052
	Jan. 3– 4*	730	1.013	0.695	1.224	0.028	0.104	1.555	36.8	36.8	0.60	2.48	0.077	0.089
	Jan. 4– 5	736	1.009	0.576	1.147	0.019	0.105	1.381	34.3	38.8	0.47	2.81	0.076	0.072
	Jan. 5– 6	750	1.010	0.836	0.944	0.214	0.067	1.48	46.5	29.8	4.8	1.68	0.057	0.066
	Jan 6– 7*	844	1.009	0.689	1.569	0.065	0.063	1.63	34.8	45.0	1.32	1.44	0.061	0.063
	Jan. 7– 8	820	1.009	0.641	1.524	Very low	0.080	1.53	34.6	46.6	Very low	1.94	0.050	0.072
	Jan. 8– 9	780	1.009	0.370	1.729	Very low	0.111	1.49	19.4	54.3	Very low	2.77	0.052	0.071
	Jan. 9–10	724	1.010	0.418	1.085	0.062	0.037	1.287	26.8	39.4	1.59	1.06	0.049	0.067
	Jan. 10–11*	710	1.012	0.362	1.658	0.082	0.056	1.5	19.9	51.7	1.81	1.38	0.060	0 072
	Jan. 14–15*	930	1.008	0.50	0.837	0.007	Very low	1.21	34.0	32.3	0.2	Very low	0.068	0.058
	Jan. 15–16	1,320	1.008	0.718	1.187	0.009	0.056	1.756	33.7	31.5	0.2	1.2	0.091	0.085
	Jan. 16–17	646	1.010	0.712	0.968	0 006	0.082	1.43	41.0	31.6	2.24	2.12	0.063	0.064
	Jan. 17–18	880	1.010	0.790	1.372	0.009	0.085	1.774	36.6	36.1	0.2	1.78	0.065	0.085
	Jan. 18–19*	750	1.009	0.678	1.393	0 011	0.095	1.596	35.0	40.8	0.23	2.2	0.058	0.071
	Jan. 19–20	766	1.010	0.458	1.376	0.025	0.091	1.416	26.6	45.4	0.58	2.4	0.049	0.074
	Jan 20–21	816	1.009	0.483	1.173	Low	0.092	1.28	31.1	42.8	Low	2.67	0.048	0.054
	Jan. 21–22	960	1.010	0.555	1.438	Low	0.120	1.532	29.8	43.9	Low	2.9	0.060	0.076
	Jan. 22–23	740	1.008	0.367	1.196	Low	0.087	1.202	25.1	46.5	Low	2.7	0.049	0.061
	Jan. 23–24	1,010	1.008	0.453	1.573	Low	0.091	1.456	25.6	50.5	Low	2.3	0.036	.....
H. C., 6 mos.; 11 lbs.; (no eczema); urine	Jan. 27–29*	622	1.011	0.612	4.548	Low	0.0248	2.935	17.2	72.4	Low	0.31	0.0337	0.184
	Jan. 28–29	580	1.014	1.475	3.649	0.0125	0.0156	3.28	37.0	52.0	0.13	0.18	0.0217	0.209
	Jan. 29–30	418	1.014	1.106	4.008	0.0087	0.0104	2.926	31.1	64.0	0.10	0.13	.....	.....
	Jan. 30–31*	362	1.015	1.044	3.426	0.010	Low	2.914	29.5	54.9	0.11	Low	.....	.....
	Jan. 31–Feb. 1	416	1.013	0.795	1.646	.....	0.0208	3.098	21.1	24.8?	....	0.35	0.0108	0.203
	Feb. 1–2	526	1.014	1.592	3.719	0.0877	Low	3.49	37.6	49.8	0.84	Low	0.0123	.....
	Feb. 2–3	410	1.012	0.611	3.242	0.0672	Low	2.296	21.9	66.0	0.97	Low	0.0107	.....
	Feb. 3–4*	440	1.012	0.934	3.137	0.0571	0.0262	2.501	30.8	58.6	0.76	0.39	.....	0.161
	Feb. 4–5	360	1.015	1.322	3.559	0.0515	Low	3.004	36.3	55.4	0.57	Low	0.0073	0.182
	Feb. 5–6	330	1.015	0.871	3.441	0.075	Low	2.616	27.4	61.5	0.95	Low	0.0101	0.153
	Feb. 6–7	410	1.014	0.898	4.004	0.0707	0.0162	3.065	24.1	61.0	0.77	0.20	0.0201	0.193
	Feb 7–8	424	1.017	1.124	4.674	0.0432	.....	3.645	25.4	60.0	0.4	.. .	.....	.....

* Sugar increased in diet.

TABLE 10.—*Analysis of Twenty-Four Hour Specimens of Stools*

	Date, 1921	Weight, Dried	Nitrogen, Gm.	Nitrogen, Percentage	Ash, Gm.	Ash, Percentage	Calcium Monoxid, Gm.	Calcium Monoxid, Percentage	Calcium Monoxid, Percentage of Ash	Fat, Gm.	Fat, Percentage	Carbohydrate, Gm. (Glucose)
P. S. Normal stools; feces	Nov. 9–10	4.903	0.182	3.7	1.259	25.7	0.886	18.1	70.4	0.557	11.4	0.432
	Nov. 10–11	8.412	0.288	3.4	2.278	27.1	1.724	20.5	75.7	2.04	24.2	1.038
	Nov. 11–12	9.236	0.354	3.8	2.343	25.1	1.478	16.0	63.1	1.912	20.7	1.504
	Nov. 12–13	4.779	0.135	2.8	1.094	22.9	0.731	15.3	66.9	1.120	23.4	1.001
	Nov. 13–14	18.465	0.720	3.9	3.988	21.6	2.792	15.1	70.0	4.254	23.0	.....
	Nov. 14–15	.....	.....	....	.....	....	.....	....	....	3.675	19.9	3.752
	Nov. 15–16	23.537	0.927	3.9	5.484	23.3	3.750	16.0	68.5	4.39	18.6	5.279
	Nov. 16–17	8.313	0.323	3.9	1.675	20.2	1.114	13.4	66.4	1.272	15.3	1.97
	Nov. 17–18	10.941	0.448	4.1	2.588	23.7	1.744	15.9	67.4	1.718	15 7	2.035
	Nov. 18–19	7.927	0.332	4.2	1.990	25.1	1.402	18.4	73.6	1.110	14.0	1.374
	Average.....	.....	.....	3.9	.....	23.8	.....	....	....	.....	18.3	
W. K. Normal stools	Nov. 20–21	3.003	.....	....	0.818	27.2	0.622	20.7	76.0	0.634	23.1	0.250
	Nov. 21–22	2.140	.....	....	.....	....	.....	....	....	0.753	35.2	0.078
	Nov. 22–23	3.733	.....	....	1.224	32.8	0.974	26.1	79.5	0.783	21.0	0.164
	Nov. 23–24	6.429	0.303	4.72	1.167	18.1	0.926	14.4	79.5	2.083	32.4	0.288
	Nov. 24–25	4.758	0.206	4.32	1.323	27.8	0.959	20.2	72.5	1.542	32.4	6.210
	Nov. 25–26	6.617	0.337	5.1	1.757	26.5	1.235	18.7	70.3	2.044	30.8	0.220
	Nov. 26–27	7.563	0.367	4.85	1.701	22.5	1.291	17.1	75.9	2.082	27.8	0.828
	Nov. 27–28	6.595	0.333	5.05	1.570	23.8	1.232	18.7	78.5	2.170	32.9	0.476
	Nov. 28–29	6.573	0.349	5.3	1.524	23.2	1.111	16.9	72.8	1.860	28.2	0.438
	Nov. 29–30	5.895	0.309	5.3	1.309	20.2	0.894	15.2	68.2	2.010	34.1	0.252
	Average.....	.....	.....	4.9	.....	24.9	.....	....	....	.....	29.8	
M. T. Constipation	Dec. 27–28	7.991	0.328	4.1	1.674	20.9	1.095	13.7	65.5	....	....	0.263
	Dec. 28–29	15.546	0.440	2.8	2.818	18.1	1.685	10.8	59.8	.....	.. .	0.513
	Dec. 29–30†											
	Dec. 30–31	15.947	0.490	3.1	2.641	16.5	1.172	7.4	44.4	.. ..	....	1.442
	Dec. 31 to Jan. 1, 1922†											
	Jan. 1– 2†											
	Jan. 2– 3†											
	Jan. 3– 4	22.944	0.888	3.9	4.290	18.7	2.707	11.8	63.3	.. ..	....	1.753
	Jan. 4– 5†											
	Jan. 5– 6	18.828	0.830	4.4	2.538	13.5	1.337	7.1	52.7	.....	....	2.043
	Jan. 6– 7†											
	Jan. 7– 8†											
	Jan. 8– 9	9.215	0.419	4.5	1.628	17.7	0.981	10.6	60.2	2.156	23.4	0.427
	Jan. 9–10†											
	Jan. 10–11*	9.603	0.401	4 2	1.745	18.2	1.028	10.7	58.9	1.875	19.5	0.824
	Jan. 11–12	10.797	0.564	5.2	1.773	16.4	1.166	10.8	65.8	1.868	17.3	0.789
	Jan. 12–13†											
	Jan. 13–14	10.348	0.534	5.2	1.546	14.9	0.792	7.6	51.2	1.728	16.7	0.989
	Jan. 14–15*	7.036	0.398	5.7	0.645	9.2	0.381	5.4	59.2	0.978	13.9	2.322
	Jan. 15–16†											
	Jan. 16–17	9.916	0.494	5.0	1.356	13.7	0.916	9.2	67.5	2.460	24.8	1.389
	Jan. 17–18‡											
	Jan. 18–19*†											
	Jan. 18–20†											
	Jan. 20–21	13.140	0.648	4.9	1.934	14.7	1.302	9.9	67.3	2.338	17.8	1.007
	Jan. 21–22†											
	Average.....	.....	.....	4.4	.....	16.0	.....	....	....	.....	19.0	

* Sugar increased in diet.
† No stools.
‡ Specimen thrown out by porter.

The body medication remained the same throughout, but for the face milk of magnesia was substituted. In spite of the formula change, the face became worse than it had ever been, and it was not until a White's crude coal tar ointment, ammoniated mercury 3 per cent., Sulpho-ichthyolate preparation 2 per cent. in starch and zinc was applied that any improvement was noticed—fully two weeks after the fat was decreased.

Therefore a summary of the facts mentioned above tends to show that the increase in the fat had nothing to do with the eczematous condition.

1. The infant on the lower fat diet did not tend to improve.

2. On the higher fat diet there was steady improvement for almost two weeks, besides maintenance of weight.

3. When the patient became worse and the formula was changed ot the lower fat content, the condition did not improve until two weeks after various medications had been applied.

METHODS

The chemical methods of analysis and manner of tabulation of results were the same as those used in the other cases.

DISCUSSION OF RESULTS OF E. D.

Before the fat was increased, the percentage of dried weight of stool was 30.5; after the fat was increased, the average was 39.8 per cent. or 38.7 per cent.; and 41 per cent. when 20 ounces or 22 ounces of the top half of the milk was used. Averaging figures on all diets, 34.1 per cent. is obtained, which is almost similar to the 36.1 per cent. given by Courtney and Fales. Forty-four and nine-tenths per cent. were obtained in the previous case of S. H.

The following table shows some individual cases as reported by them, with which the present case is compared.

TABLE 11.—*Table from Article of Courtney and Fales, Am. J. Dis. Child., Vol. 17, 1919*

Fat Intake, Gm.	Fat Percentage Dried Weight Stool
22.0	25.7
30.5	51.0
36.5	36.4
Case of S. H.	
20.5	24.6
34.4	42.2
34.9	50.7
35.1	62.2
Average	44.9
Case of E. D.	
22.7	30.5
39.7	38.7
39.9	41.0
22.7	26.1
Average	34.1

Since the constituents of the diet were not weighed, the fat intake could only be obtained approximately, yet in spite of this the similarity with other individual cases previously reported is close. Agreeing with them, there seems to be no definite relationship between the daily fat intake and percentage of fat in the stool.

In regard to the percentage of retention of fat, the following results can be compared.

TABLE 12.—*Table from Article of Courtney and Fales, Am. J. Dis. Child., Vol. 17, 1919*

Fat Intake, Gm. Daily	Fat Output, Gm. Daily	Fat Retained, Gm. Daily	Percentage Retained
14.6 - 36.5	2.33	24.4	91.3

TABLE 13.—*Case of E. D.*

	Fat Intake, Gm. Daily	Fat Output, Gm. Daily	Fat Retained, Gm. Daily	Percentage Retained
	22.7	2.098	20.59	90.7
	37.7	3.349	36.14	89.4
	39.9	3.864	36.15	90.4
Average	34.4	3.103	30.96	90.1
		Average of Cases E. D. and S. H.		
S. H. ...	31.2	2.87	28.3	90.5
E. D. ...	34.4	3.103	30.96	90.1
Average	32.8	2.990	29.63	90.3

TABLE 14.—*Relation of Calcium Absorption to Calcium Intake Per Kilogram*

Calcium Oxid Intake, Gm. Daily	Calcium Oxid Output, Gm. Daily	Calcium Oxid Retention, Gm. Daily	Calcium Oxid Retention, Percentage	Calcium Oxid Intake, per Kilogram	Calcium Oxid Retention, Gm. per Kilogram
0.951	0.783	0.168	16.6	0.18	0.022
0.476	0.853	— Bal.	— Bal.	0.091	— Bal.
0.951	0.950	+ Bal.	+ Bal.	+ Bal.	+ Bal.
Average, Courtney and Fales *	0.70	.....	35-55	.....	0.089
		Case of S. H.			
1.307	0.957	0.35	26.7	0.21	0.057
0.769	0.691	0.078	10.1	0.13	0.013

* Am. J. Dis. Child., vol. 19, 1920.

Courtney and Fales give as a retention range from 85.8 to 97.4 per cent. By consulting Table 15, one can readily see that all results fall within these figures, with the exception of those obtained on two days, one being 80.8 and the other 83.3. It is noteworthy but probably without significance that these lower values were obtained on the first day in each instance, when the formula was changed. The same circumstance happened also in the previous case (Table 6 in report on S. H.). This was probably due to the mechanism trying to adjust itself to the change. Agreeing with Courtney and Fales, there seems to be no strik-

ing relationship between the fat intake and the percentage of fat retained. They found that it happened only when the intake was abnormally low.

By comparing the estimates in Table 14 with normal figures, one can see that the percentage of calcium absorbed in both cases is lower than the average for normal children, and seems to be similar to figures given by Courtney and Fales in the case of rachitic infants.

TABLE 15.—*Fat Metabolism of E. D.*

Date, 1923	Fat Intake, Gm.	Fat Output, Gm.	Fat Retention, Gm. Daily	Percentage Retention
April 23-24	22.7	1.326	2.137	94.1
April 25-26	22.7	2.120	20.58	90.6
April 26-27	22.7	2.727	19.97	87.9
April 27-28	22.7	1.346	21.35	94.0
April 28-29	39.7	4.231	35.46	80.0
April 29-30	39.7	3.442	36.25	91.9
May 1- 2	39.7	3.440	35.26	88.8
May 2- 3	39.7	2.236	37.46	94.4
May 3- 4	39.7	3.398	36.30	91.4
May 4- 5	39.9	3.084	36.81	92.2
May 5- 7	39.9	4.895	35.00	87.7
May 7- 8	39.9	3.573	36.32	91.4
May 8- 9	39.9	3.904	35.99	90.3
May 17-18	22.7	3.761	18.93	83.3
May 19-20	22.7	1.630	21.07	92.8
May 20-21	22.7	1.982	20.71	91.2
May 21-22	22.7	1.892	20.80	91.6

The average ash percentage of dried weight of stool, as given by Courtney and Fales for infants on modification of cow's milk, is 24.1 per cent.; the average in this investigation is 15.7 per cent., which is considerably lower.

TABLE 16.—*Calcium Excretion in Relation to Calcium and Fat Intake*

Calcium Oxid Intake, Gm. Daily	Fat Intake, Gm. Daily	Calcium Oxid Output, Gm. Daily	Fat Output, Gm. Daily
0.951	22.7	0.783	2.098
0.476	38.8	0.853	3.606
	Case of S. H.		
1.307	20.5	0.957	1.38
0.769	34.8	0.697	3.36

Agreeing with Courtney and Fales, as with the previous case of S. H., there seems to be no constant relationship between the amount of fat intake and the calcium excreted. However, in this case of E. D., when the fat is increased, a wide negative balance in calcium metabolism is obtained. However, the evidence is not weighty enough to point to the fat as being the cause, since when the formula was changed to the lower fat content only a slight balance over equilibrium was obtained.

The average percentage of nitrogen of dried weight of stool is 1.94 per cent., which is lower than the figure given by Courtney and Fales for infants on a diet of modification of cow's milk.

TABLE 17.—*Analysis of Twenty-Four Hour Specimens of Urine of E. D. Aged 3 Months*

Date, 1923	Quantity, C.c.	Specific Gravity	Ammonia, Gm.	Urea, Gm.	Uric Acid, Gm.	Creatinin, Gm.	Total Nitrogen, Gm.	Ammonia, Nitrogen, Total Percentage	Urea Nitrogen, Total Percentage	Uric Acid Nitrogen, Total Percentage	Creatinin Nitrogen, Total Percentage	Sugar (Gluc.), Gm.	Sulphate Total
April 23–24	130	1.014	0.017	1.44	0.065	0.007	1.509	1.0	30.0	1.43	1.16	.....	0.042
April 25–26	445	1.020	0.061	1.008	0.018	0.025	1.091	4.2	45.6	5.55	0.94	0.108	0.030
**													
April 27–28	125	1.014	0.042	0.839	0.074	0.006	0.622	5.6	63.0	3.96	0.36	0.021	.....
April 28–29	330	1.006	0.120	2.136	0.014	0.012	1.109	10.0	79.9	4.20	0.42	0.059	0.048
April 29–30	260	1.010	0.115	1.432	0.086	0.010	1.134	8.3	58.0	2.52	0.33	0.045	0.135
May 1– 2	320	1.009	0.131	1.724	0.010	0.011	1.101	10.0	48.0	3.02	0.36	0.080	0.052
May 2– 3	250	1.010	0.143	1.241	0.075	Low	0.739	14.9	79.4	3.42	Low	0.058	0.032
**													
May 3– 4	350	1.010	0.250	1.112	0.021	Low	1.092	17.3	79.4	6.40	Low	0.145	0.032
May 4– 5	315	1.011	0.207	1.658	0.015	Low	1.234	12.8	62.7	4.04	Low	.....	0.052
May 6– 7	250	1.011	0.285	0.803	0.050	0.010	0.945	15.6	36.6	1.05	0.39	0.008	0.048
May 7– 8	150	1.009	0.124	0.719	0.032	Low	0.520	19.2	64.6	2.04	Low	0.035	0.016
***													
May 8– 9	200	1.010	.....	.....	0.071	Low	0.627	....	....	3.71	Low	0.066	0.049
May 17–18	360	1.011	0.200	1.850	0.021	0.008	1.44	10.5	53.5	4.85	0.20	0.083	0.089
May 18–19	500	1.010	0.268	.....	0.037	Low	1.554	13.1	....	7.23	Low	0.101	0.107
May 19–20	285	1.009	0.385	.....	0.094	0.012	0.878	33.5	....	3.56	0.51	0.045	0.042
May 20–21	410	1.100	0.487	0.656	0.010	0.014	1.15	26.2	26.7	2.89	0.32	0.066	0.077
May 21–22	170	1.012	0.161	0.682	0.056	Low	0.522	23.5	57.4	3.57	Low	Low	0.037
May 22–23	225	1.012	0.211	0.510	0.075	0.005	0.540	29.8	44.0	4.63	Low	0.025	0.049

** Fat increased in diet.
*** Fat decreased in diet.

TABLE 18.—*Analysis of Twenty-Four Hour Specimens of Stools of E. D. Aged 3 Months*

Date, 1923	Weight, Dried, Gm.	Nitrogen, Gm.	Nitrogen, Percentage	Ash, Gm.	Ash, Percentage	Calcium Oxid, Gm.	Calcium Oxid, Percentage	Calcium Oxid, Percentage Ash	Fat, Gm.	Fat, Percentage	Carbohydrate (Glucose), Gm.
April 23–24	4.020	.....	....	0.804	20.0	0.406	10.1	50.4	1.326	33.0	0.206
April 25–26	6.625	0.148	2.24	1.378	20.8	0.928	14.0	66.5	2.120	32.0	0.210
April 26–27	7.140	0.179	2.52	1.071	15.0	0.984	13.5	90.0	2.727	38.2	0.535
**											
April 27–28	7.923	0.166	2.10	1.426	18.9	0.832	10.5	58.3	1.346	17.0	.....
April 28–29	10.295	1.461	1.42	1.842	17.9	1.235	12.0	67.0	4.231	41.1	....
April 29–30	7.567	0.143	1.89	1.135	15.0	0.681	9.0	60.0	3.442	45.5	0.378
April 30–May 1	7.792	0.130	1.68	1.238	15.9	0.857	11.0	69.2	.....	....	0.257
May 1– 2	9.556	.....	....	1.290	13.5	1.146	12.0	88.0	3.440	36.0	0.353
May 2– 3	7.530	0.149	1.96	0.904	12.0	0.843	11.2	93.1	2.236	29.7	0.251
**											
May 3– 4	8.190	.....	....	1.040	12.7	0.745	9.1	71.6	3.398	41.5	0.262
May 4– 5	9.100	0.165	1.82	1.319	14.5	0.737	8.1	55.1	3.084	33.9	0.182
May 5– 7	11.655	0.136	1.17	1.45	12.5	0.967	8.3	66.4	4.895	42.0	0.215
May 7– 8	6.770	0.104	1.54	0.947	14.0	0.670	9.9	60.2	3.573	42.1	.....
***											
May 8– 9	8.487	0.213	2.52	1.196	14.1	0.779	9.2	65.1	3.904	46.0	0.432
May 17–18	8.341	0.210	2.52	1.406	16.9	0.917	11.0	65.0	3.761	45.1	0.502
May 19–20	6.522	0.109	1.68	....	....	....	....	....	1.630	25.0	0.251
May 20–21	7.020	0.157	2.24	1.406	20.0	0.984	14.0	69.9	1.982	28.2	0.260

** Fat increase in diet.
*** Fat decrease in diet.

As to the increase in fat and the amount of nitrogen excreted in the form of ammonia, the figures show the following results: Before the increase, the average value of ammonia nitrogen was 2.6 per cent. of the total nitrogen, which is within normal limits. After the increase in the fat, the value was 12.6 per cent. However, when the fat was decreased, the value became 22.7 per cent. Looking at only the first two values, one might conclude that the fat was responsible for the increase in the ammonia nitrogen, if it were not for the fact that in spite of the fat being reduced, the excretion not only was abnormal, but increased 100 per cent. This circumstance tends to show that the fat had nothing to do with the increase in the ammonia nitrogen excretion. Moreover, regardless of the increase and decrease in the fat, the urine was normal in reaction for ten days, while the remaining time of the experiment it was alkaline. The average value for uric acid nitrogen, 3.78 per cent., is somewhat higher than the normal, and the creatinin nitrogen, 0.39 per cent., is much lower, some days being so low that colors could not be matched in the colorimeter.

CONCLUSIONS AND SUMMARY: CASE E. D.

In this case of infantile eczema, the following conclusions can be drawn regarding metabolism:

1. The amount of ammonia nitrogen excreted is abnormal, but as in the previous case, there seems to be no definite relationship between this excretion and the fat increase in the diet (Table 17).

2. The fat metabolism appears to be normal: the percentage retained, 90.1, compares very well with Courtney and Fales' figure of 91.3 as also the previous case of S. H., 90.5 per cent. (Tables 6 and 18).

3. The average percentage of nitrogen in the stool, 1.94 per cent., is somewhat lower than the figure given by Courtney and Fales, as also that obtained in the previous case (Table 17).

4. Agreeing with the previous case, the percentage of calcium absorbed is lower than the average of 0.089 gm. per kilogram, and the excretion is higher but similar to the figure of 0.86 given by Courtney and Fales in the case of rachitic infants (Tables 14, 16, 18).

5. Agreeing with Courtney and Fales, as in the previous case, there seems to be no constant relationship between the amount of fat intake and the calcium excreted. However, in the case of E. D., when the fat is increased, a wide negative balance in calcium metabolism is obtained. However, the evidence is not weighty enough to point to the fat as being the cause, since when the formula was changed to the lower fat content, only a slight balance over equilibrium was obtained (Table 16).

6. Although unlike S. H., E. D. did not recover as he did after the increase of the fat, there are certain facts which tend to show that even in this case the fat had nothing to do with the eczematous condition:

1. On the lower fat diet, the infant did not tend to improve.

2. On the higher fat diet, there was steady improvement for almost two weeks, besides maintenance of weight.

3. When the patient became worse and the formula was changed to the lower fat content, the condition did not improve until after two weeks, after various medications had been applied.

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A COMPARATIVE STUDY OF THE HERROLD PRECIPITATION TEST AND THE WASSERMANN REACTION

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Since the advent of the precipitation tests for syphilis, several papers have appeared giving the results of the various modifications of the test as compared with the Wassermann test.

A clinical study of a sufficient number of cases tested by one of the new methods and checked by a routine Wassermann test should be of value. This was accomplished by using the routine Wassermann test at the Central Free Dispensary from Nov. 27, 1922, to July 26, 1923. In the study of this wide range of material, certain principles of value were established.

The Wassermann test was run with a cholesterolized antigen and an acetone-insoluble antigen. Only three degrees of hemolysis are recorded: (1) complete inhibition of hemolysis + + +, (2) moderate inhibition of hemolysis + +, (3) slight inhibition of hemolysis +.

The Herrold[1] modification was adopted as a precipitation test. We used the alcoholic extract of beef heart after a preliminary ether extraction, described by Neyman and Gager.[2] This was reinforced with 0.4 per cent. cholesterol. Dilutions were made by placing a small, measured amount of salt solution in a Wasserman tube and rapidly blowing in the proper amount of extract with a 1 c.c. pipet graduated to hundredths. Using this method, no secondary dilution was necessary. After a large series of titrations, we found that the proportion of one volume of extract to sixteen volumes of saline gave the greatest degree of accuracy. Only inactivated serum was used as a routine. A normal salt control was run with each serum.

The Herrold modification was adopted on account of its simplicity and ease of reading. As in all precipitation tests, certain technical details are of extreme importance:

(*a*) All glassware and pipets must be scrupulously clean and absolutely sterile.

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1. Herrold, Russell D.: A Ring or Contact Precipitation Test for Syphilis, J. A. M. A. **79**:957 (Sept. 16) 1922.

2. Neyman, C. A., and Gager, L. T.: A New Method for Making Wassermann Antigen from Normal Heart Tissue, J. Immunol. **2**:573 (Oct.) 1917.

(*b*) The salt solution must be sterile and freshly filtered before using. The presence of small particles is deleterious.

(*c*) The extract dilution is prepared fresh each day and stands one hour before using.

(*d*) The capillary pipets are drawn to an extremely fine bore before using to layer the emulsion under the serum. Otherwise a mixture rather than a layer results.

(*e*) The method of preparing the serum is important. The blood should be drawn several hours after a meal to avoid lipemic serum. It should be drawn with a dry, sterile syringe or drawn directly into a dry sterile tube. It must be kept sterile. The proper separation of the clot is important.

With clear serums, freshly prepared, there is possible a higher degree of correspondence with the Wassermann reaction than is reported in this series. Many of these serums were taken by students and allowed to stand several hours in a warm room. It was impossible in many cases to draw the blood several hours after a meal to avoid lipemic serums.

In the series to be described, 2,025 tests were run. For convenience, these have been classified clinically under three main headings: (*a*) syphilitic, (*b*) nonsyphilitic, and (*c*) cases in which no history was obtainable. To keep these statistics within reasonable bounds all tests have been grouped as (1) strong, two plus or three plus, (2) weak, one plus, and (3) negative.

Two groups of agreement were defined: (1) virtual agreement, serums in which both the Wassermann and the precipitin reactions were both strong, both weak, or both negative; and (2) approximate agreement: serums in which both tests were strong or both tests were weak, or one test was weak ($\pm$) and the other negative.

A. SYPHILITIC CASES

A total of 1,075 cases of syphilis were tested, with virtual agreement in 865 cases, or 80.4 per cent., and approximate agreement in 946 cases, or 88 per cent. When the two tests did not agree, the Wassermann test was the more sensitive in the majority of cases.

For more detailed analysis, the syphilitic cases were considered under the following classifications: (1) chancre with no secondary manifestations; (2) syphilis with a history of secondary lesions; (3) late cutaneous syphilids; (4) late visceral and bone lesions; (5) cases of women with a history of miscarriages, stillbirths, or those cases in which the husbands or children were syphilitic; (6) congenital syphilis; (7) latent syphilis; (8) tabes; (9) general paresis, and (10) cerebrospinal syphilis.

TABLE 1.—*Results of Wassermann and Herrold Tests in Treated and Untreated Syphilis*

	Treated Cases							Untreated Cases						
Classification	Cases	Virtual Agreement	Per Cent.	Agree Except ±	Per Cent.	Wassermann ++ or +++ Herrold ±, per Cent.	Herrold ++ or +++ Wassermann ±, per Cent.	Cases	Virtual Agreement	Per Cent.	Agree Except ±	Per Cent.	Wassermann ++ or +++ Herrold ±, per Cent.	Herrold ++ or +++ Wassermann ±, per Cent.
Chancre with no secondary manifestations	...	...	....	...	....	....	....	43	33	76.7	35	81.3	18.6	0
Syphilis with a history of secondary manifestations	...	...	....	...	....	....	....	81	81	100.0				
Treated early syphilis.	260	197	75.8	230	88.4	10.4	1.1							
Treated late syphilis...	90	65	72.7	74	82.2	12.2	5.5							
Total treated syphilis.	350	262	74.8	304	86.8									
Cutaneous syphilitic cases	28	19	67.8	22	79.5	21.4	0	39	34	87.1	35	89.6	7.7	2.5
Late visceral and bone lesions	61	51	83.6	55	90.1	4.9	4.9	53	48	90.6	48	90.6	5.6	3.8
Women with suggestive history	45	37	82.2	40	88.8	8.9	2.2	57	52	91.2	54	94.6	3.5	1.7
Congenital syphilis....	43	38	88.4	41	95.4	4.6	0	36	36	100.0				
Latent syphilis.....	44	33	75.0	37	84.0	6.8	9.0	38	25	65.8	26	68.4	23.7	7.9
Tabes.....	46	33	71.6	37	80.3	17.4	2.2	12	12	100.0				
General paresis.....	8	8	100.0	0	0	0	0	6	4	66.6	4	66.6	33.3	0
Cerebrospinal syphilis.	63	43	68.3	53	84.2	12.7	3.2	20	16	80.0	18	90.0	5.0	5.0
All central nervous system syphilis.....	117	84	71.8	98	83.8	13.7	2.5	38	32	84.2	34	90.0	8.0	2.6
Total all syphilis.....	688	524	71.1	597	87.2	16.5	2.8	387	341	88.1	349	90.2	7.7	2.0

It was our experience that in cases of early, primary syphilis, in which the chancre had been present but a short time, the Wassermann test became positive before the Herrold test. In a total of forty-three cases with chancre and no secondary manifestations, thirty-three cases were in agreement, or 76.7 per cent. In the group showing disagreement the Wassermann test was three plus and the Herrold negative in two cases, or 4.6 per cent. The Wassermann test was two plus and the Herrold negative in four cases, or 9.2 per cent. The Wassermann test was three plus and the Herrold one plus in one case, or 2.3 per cent. In no case was the Herrold test more sensitive than the Wassermann test.

In 260 cases with a history of secondary manifestations and early treatment there was closer agreement between the two tests than in ninety similar cases in which late treatment was received. In both groups the Wassermann test was markedly more sensitive than the precipitin test. However, in 3.4 per cent. of all treated cases the Herrold test was appreciably the more delicate test. In eighty-one cases of syphilis with active secondary skin manifestations, in which the patients had received no treatment, there was virtual agreement in 100 per cent.

In sixty-seven cases of late cutaneous lesions of syphilis, the Wassermann test proved to be more sensitive than the precipitin test, particularly in the cases in which the patients had received treatment. Nevertheless, in a few instances in which treatment had not been given, the precipitin test proved useful. It is interesting to note in this connection that in five of the cases, or 12.5 per cent., that showed clinically late syphilitic skin lesions without previous treatment, both tests were negative. These cases all responded to antisyphilitic therapy.

In sixty-one cases of treated late visceral and bone manifestations of syphilis and in fifty-three cases in which the patients were untreated, there was approximate agreement between the two tests in about 90 per cent. In the cases in which the two tests did not agree, the Wassermann test was somewhat more sensitive in the untreated cases.

Thirty-six cases of untreated congenital syphilis gave 100 per cent. agreement between the two tests, but the Wassermann test was more delicate in the forty-three cases in which the patients had received treatment.

There was marked divergence between the two tests in cases of latent syphilis, especially in those cases in which the patient had received no treatment. Of a total of thirty-eight untreated patients, 23.7 per cent. had a strongly positive Wassermann and a weak or negative Herrold test. However, in 7.4 per cent., the opposite was true, and the Herrold was the more delicate test.

In the central nervous system group we were struck by the relatively large number of cases of tabes in which the Wassermann test was strongly positive and the precipitin test negative. This happened in four cases (8.7 per cent.) of tabes in which treatment had been given in a total of forty-six cases. In two cases, or 4.3 per cent., the Wassermann test was two plus and the Herrold test negative. In only one case was the Wassermann test negative and the Herrold test two plus. In one case of general paresis with positive spinal fluid findings, the Wassermann test was three plus and the Herrold test negative on repeated examinations. Out of a total of sixty-three cases of cerebrospinal syphilis in which treatment had been given, three cases, or 4.7 per cent., gave a three plus Wassermann test and a negative Herrold test. Five cases, or 8 per cent., gave a two plus Wassermann test and a negative Herrold test. In general, it is evident that in a large percentage of cases of central nervous system syphilis, the precipitin test is unreliable.

TABLE 2.—*Complete Summary of all Cases Tested*

Classification	Cases	Agreement	Per Cent.	Agree Except ±	Per Cent.	Wassermann ++ or +++ Herrold ±, per Cent.	Herrold ++ or +++ Wassermann ±, per Cent.
Syphilitic	1,075	865	80.4	946	88.0	9.5	2.5
Nonsyphilitic	885	802	90.6	864	97.6	1.7	0.68
No history obtainable	65	55	84.7	56	86.2	4.6	9.2
Total	2,025	1,722	85.0	1,866	92.0		

B. NONSYPHILITIC CASES

This classification included cases studied at the Central Free Dispensary in the department of general medicine, pediatrics, neurology, orthogenics, urology, gynecology, obstetrics and dermatology. We were particularly interested in this group of cases, for if the precipitin test is not specific, it is valueless. The work was done in a frank spirit of skepticism. We believe that it represents the limitations of the test as ordinarily performed. Of a total of 885 cases, there was virtual agreement in 802 cases, or 90.6 per cent., and approximate agreement in 864 cases, or 97.6 per cent. There was a false strong Herrold test in only 0.68 per cent. This is a satisfactory showing, as the percentage of false positive precipitin tests is less than that of the Wassermann tests in this series.

C. CASES IN WHICH NO HISTORY WAS OBTAINABLE

There were sixty-five cases in this group. Most of these patients made only one visit to the dispensary. There was considerable variation between the tests, but it was impossible to determine which was the more accurate.

CONCLUSIONS

1. The Herrold precipitin test is specific.

2. It agrees with the Wassermann test in untreated secondary syphilis and in untreated congenital syphilis.

3. It becomes positive later than the Wassermann test in untreated primary syphilis.

4. It is less sensitive than the Wassermann test in treated cases and later in the disease.

5. It is unsatisfactory in syphilis of the central nervous system.

6. It is positive in a small but definite number of cases of syphilis in which the Wassermann test is negative.

7. It is liable to errors of a different order than those of the Wassermann test.

8. It is a valuable control for routine use with the Wassermann test.

9. It is unsatisfactory with lipemic serums.

10. If skilfully used, it is an admirable test for office use.

AN ORIGINAL BACTERIOLOGIC RESEARCH ON PRURITUS OF THE PERINEUM

(PRURITUS ANI, VULVAE AND SCROTI) *

J. F. MONTAGUE, M.D.

NEW YORK

Of the many theories which have from time to time been offered in an attempt at explanation of this malady, none has been more alluring than that it is due to bacterial invasion. This possibility was first asserted by Murray, who believed he had found a specific causative organism—a strain of streptococcus which is known to bacteriologists as *Streptococcus fecalis*. For some years he contended that here was the long sought for primary etiologic factor, and by his persistent reports he won many adherents to his views. At present this theory has been largely discarded, partly because of lack of evidence as to the pathogenicity of *Streptococcus fecalis* and partly for the reason that vaccine treatment based on the use of *Streptococcus fecalis* is totally unsatisfactory.

More recently, J. M. Winfield,[1] in presenting a review of cases concludes that 80 per cent. "had an infection of the skin covering the affected parts caused either by the colon bacillus or the Streptococcus fecalis or by both together." In 100 per cent. of these infected cases the colon bacillus "was obtained from the exudate" and in 75 per cent. *Streptococcus fecalis* was demonstrated. The same author reports eight cases in which the epidermophyton fungus was a cause.

Such a statement regarding the demonstration of infection can not be assumed on the basis of the proof offered. While I am reasonably sure, because of my studies on the pathology of the tissue in this region,[2] that infection does occur in a certain percentage of cases, my researches do not support such a statement nor does clinical experience warrant classifying four fifths of all cases as infective. The fact that colon bacilli and a strain of nonpathogenic chain growing diplococci are normal residents of the area under investigation did not seem to engage the consideration of previous investigators, nor influence their judgment as to pathogenicity.

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* Read before the American Proctologic Society at Los Angeles, California, on June 23, 1923.

1. Winfield, J. M.: The Infective Origin of Anogenital Pruritus, Arch. Dermat. & Syph. **4**:680 (Nov. 21) 1921.

2. Montague, J. F.: The Etiology and Pathogenesis of Pruritus Ani and Anal Pruritus, New York M. J. **117**: No. 8, p. 469, 1923.

With the idea, however, of ascertaining to what extent the findings of Murray and of Winfield could be corroborated, I initiated in 1918 and later in 1921 and 1922, a series of bacteriologic examinations along similar lines. The technic described in detail below was made to conform with Murray's, the only modification being an attempt to avoid contamination during the taking of the culture and a more detailed analysis of the cultures obtained.

BACTERIOLOGIC EXAMINATION

The cases subjected to this examination were in all forty-four; twenty were cases of rectal disease in which pruritus of the anus was absent and fourteen cases in which it was present without other anorectal pathology. In addition, five normal people were examined simultaneously with each group in order to control the observations. The diet in each instance was made as uniform as was possible with so many individual tastes to be considered. Full diet with meat was allowed.

In each case a culture was taken from three different regions of the posterior sector. These regions were the posterior wall of the anal canal, the posterior mucocutaneous margin and the posterior sector of the perianal region. The posterior sector was chosen because that is in many cases the seat of the most intense clinical manifestations. In all, therefore, 132 original cultures were taken for study. As an example of the manner in which each was studied, the chart of one is shown in Table 1.

The area was exposed by inserting a large sized sterile Brinkerhoff speculum with the slide posteriorly. On removing the slide the area was swabbed thoroughly with sterile liquid soap and then irrigated with sterile water. The culture was then obtained by rubbing heavily the regions indicated with sterile cotton swabs. The three swabs were then placed as obtained in separate tubes of sterile meat infusion of moderate alkalinity (0.2 to 0.5 per cent. alkaline to phenolphthalein). Incubation at 37.5 C. was allowed to proceed for twenty-four hours. At the end of this time a smear was made and stained by Gram's method. The culture was then plated out on dextrose agar. From the colony growths thus obtained one each of different types was fished out and planted on broth. From these last cultures smears were again taken, and if bacilli were found, a Hiss-Russell stab was made, whereas if cocci were found the following carbohydrate serum tubes were inoculated: inulin, raffinose, saccharose, mannite, lactose, maltose and salicin.

An elaborate tabulation might be made of this work, but the observations noted were of such a simple type that extensive tabulation would tend to confuse rather than elucidate. Instead of tabulating each case I will therefore present my findings briefly in an epitomized manner as shown in Table 2.

TABLE 1.—*Clinical Diagnosis—Pruritus Ani (Direct Pruritus of the Anus)*

PERIANAL SKIN				MUCOCUTANEOUS MARGIN		ANAL MUCOSA	
Broth				Broth		Broth	
Smear:	Plate:			Smear:	Hiss-Russell Medium	Smear:	Plate:
Gram-negative bacilli Gram-positive cocci	3 types of colonies { Smear = short thick G-rods HR medium = acid and gas			Gram-negative bacilli	Acid and gas	Gram-negative bacilli Gram-positive cocci	2 types
Dextrose Broth		Broth				Dextrose Broth	Dextrose broth
Smear:	Carbohydrate:	Smear:	Plate:			Smear:	Carbohydrate:	Smear:
Gram-positive diplococci in chains	Inulin — Raffinose — Saccharose + Mannite + Lactose + Salicin +	Clustered cocci	White elevated colonies Smear: Clustered cocci			Gram-positive cocci in chains	Inulin — Raffinose — Saccharose — Mannite + Lactose + Salicin +	Gram-negative bacilli Hiss-Russell: Acid and gas
Diagnosis:	Streptococcus nonhemolyticus III (St. fecalis)		Staphylococcus albus	B. coli	B. coli		Streptococcus nonhemolyticus I	B. coli

TABLE 2.—*Regional Bacteriologic Findings in Relation to the Presence of Pruritus*

	Anal Canal	Mucocutaneous Line	Perianal Skin
Ten normal persons	B. coli communis..... 9 Gram-positive diplococci ..... 7 B. fecalis alkaligenes..... 3 B. lactis aerogenes..... 2 B. coli communior..... 1 Anaerobes 1	B. coli communis..... 9 Gram-positive diplococci ..... 6 B. lactis aerogenes..... 2 B. fecalis alkaligenes..... 1 B. coli communior..... 1	B. coli communis..... 5 Gram-positive diplococci ..... 4 B. subtilis ..... 1 Staphylococcus epidermidis 1
Twenty with rectal disease without pruritus	B. coli communis.....19 Gram-positive diplococci .....15 B. lactis aerogenes..... 7 B. fecalis alkaligenes..... 4 Anaerobes 1	B. coli communis.....15 Gram-positive diplococci .....11 B. lactis aerogenes..... 6	B. coli communis.....11 Gram-positive diplococci ..... 7 Staphylococcus albus ..... 2 Staphylococcus epidermidis ..... 1 B. subtilis 2
Fourteen without anorectal disease other than pruritus of the anus	B. coli communis.....13 Gram-positive diplococci .....12 B. fecalis alkaligenes..... 2 B. lactis aerogenes..... 1	B. coli communis.....13 Gram-positive diplococci ..... 7 B. lactis aerogenes..... 3	B. coli communis..... 7 Gram-positive diplococci ..... 7 Staphylococcus albus 1
Summary (forty-four examined).....	B. coli communis.....41 Gram-positive diplococci .....34 B. coli communior..... 2 B. lactis aerogenes.....10 Miscellaneous 11	B. coli communis.....37 Gram-positive diplococci .....24 B. lactis aerogenes.....11 Miscellaneous 1	B. coli communis.....23 Gram-positive diplococci .....18 Staphylococcus albus ..... 3 B. subtilis ..... 3 Staphylococcus epidermidis 2

Number of original cultures taken	132
Number of growths obtained from such cultures	118
Failed to obtain growths of any kind in	14

It will be noted from the data submitted above that the bacterial species found were essentially identical in each of the three groups, and the presence or absence of pruritus of the anus apparently bore no relation to the bacterial findings. Whatever variation was noted was of such an unimportant nature as to be negligible as, for instance, the findings of *B. coli communior* in three intances, and certain differences in the occurrence of the minor components, as, for example, *B. lactis aerogenes*, etc. The term gram-positive diplococcus is used advisedly

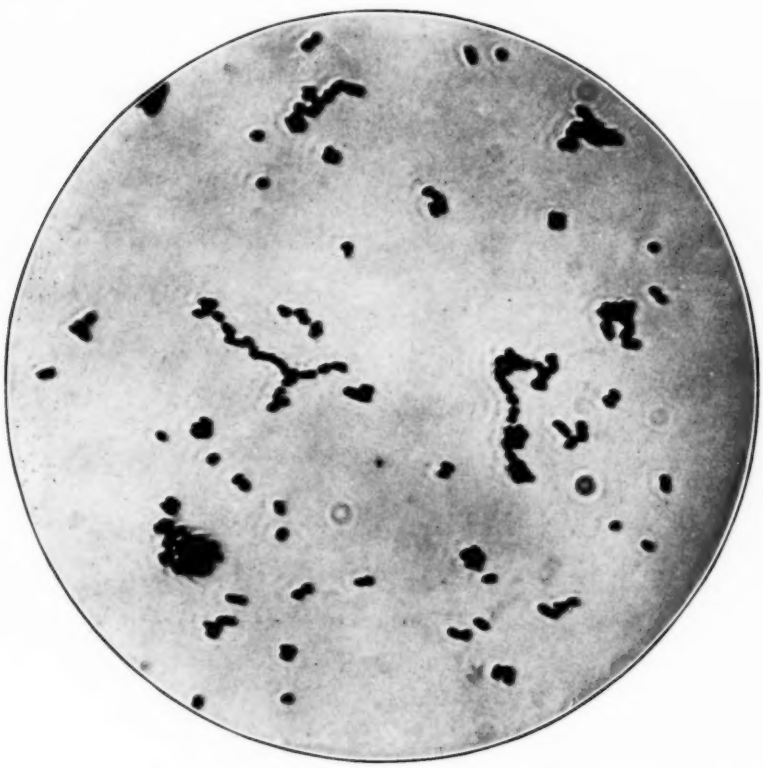

Fig. 1.—The gram-positive diplococcus which frequently grows in chains in broth mediums and is known as Streptococcus fecalis.

instead of the term streptococcus. Although some bacteriologists classify this organism as a streptococcus because of its tendency to occasional short chain formation, I prefer to refer to it according to its more usual formation, namely, as a gram-positive diplococcus, for the reason that the term "streptococcus" seems to imply pathogenic characteristics which this organism does not possess. Although it is true that this organism does assume a short chain-like form when grown in dextrose broth, this fact does not seem to me to be of impor-

tance, as many other bacteria will assume this form under the optimum conditions of a rich medium and proper temperature. It is probably the result of rapid division and subdivision of the bacteria proceeding without disturbance. The particular organism in question, moreover, shows its component diplococci markedly, even when chain form is assumed (contrast the Fig. 1 with Fig. 2). Even if we grant that it is a streptococcus by virtue of this tendency, it most assuredly does not simulate true *Streptococcus pyogenes* in any other way. To confirm the observations of other bacteriologists, and for my own satisfaction, I have injected pure cultures of this organism in enormous doses into the peritoneal cavity of guinea-pigs and into the subcutaneous tissues of dogs, without the slightest reaction or infection.

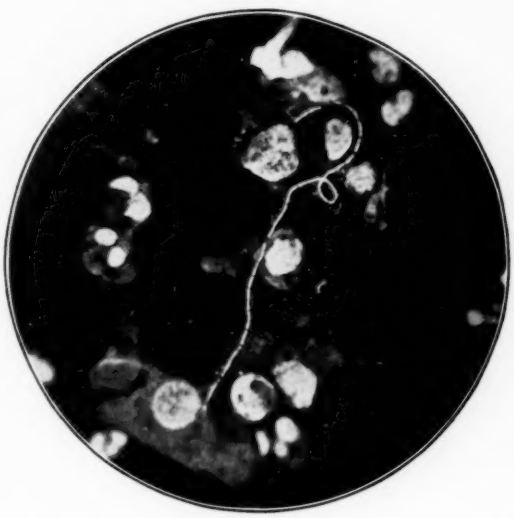

Fig. 2.—True Streptococcus pyogenes from infected tissue.

Knowing that this organism is the one that has been indicated as the specific causative agent, I determined to try to produce the disease of pruritus ani with it. However, as Park in his "Pathogenic Microorganisms" admirably states it—there is a distinction between an injection disease and an infection disease; in other words, the production of bacterial invasion by means of hypodermic injection often does not give one a true picture of the infective power of a bacterium. To meet this point, I made several small collodion sacs and placed therein pure cultures of the organism. Such collodion sacs, while not permeable to the extent of leaking, will allow dialysis to occur by the process of osmosis. I therefore concluded that if placed in the anal canal, sufficient of the culture would dialyze through to make the sac function very well as an artificial focus of infection to the neighboring tissue.

In this manner sacs containing pure cultures of the organisms in question were tried out. As a control, sacs of Ringer's solution (sterile) were used. Each sac was kept in situ for twenty-four hours, being replaced after defecation. While in place, irritation, at times itching, occurred, due no doubt, in all cases, to the resilient quality of the collodion. The intensity of irritation seemed about equal in all cases. After withdrawal, however, all patients promptly regained a normal state of sensation in the region (they were all normal cases). When, however, the perianal skin in the zone was deliberately scratched in one half of the cases and the experiment repeated, a definite pruritus remained after the removal of the sacs in several instances—one colon bacillus and two staphylococcus cases. In one of the latter the itching was so intense that I was compelled to give a full Sabouraud pastille dose of roentgen ray to eradicate the condition. The gram-positive diplococcus, which is the *Streptococcus fecalis* of Murray's experiments, showed no power to invade, although a culture made from the sac after the experiment proved its viability.

While the studies mentioned were in progress, I gradually came to the conclusion that if we were to avoid making a scientific farce out of so important a subject, the bacteriologic investigator must conduct his investigation not only along the lines of sound bacteriologic practice, but also with a thorough understanding of the underlying principles of infection and immunology.

No well trained bacteriologist would seriously consider the results of a bacteriologic examination unless he were certain that the material under investigation had been collected under such conditions and by such methods as would preclude its contamination. For instance, taking a culture of the anal mucosa or perianal skin with a nonsterile cotton swab without preliminary preparation of the area obviously renders the results valueless because we cannot rule out contaminating organisms. These were the conditions under which Murray's first experiments were performed. Nor does the preliminary scrubbing with soap and rinsing with sterile water, which method he used later and I used in my studies just described, entirely preclude the possibility of contamination, for the reason that such casual cleansing cannot be thorough to the extent of removing all organisms lodged in the folds on the surface of the anal mucosa or perianal skin. This I concluded from my series, in which after cleansing the region in a manner that I deemed thorough, I still obtained a growth in 118 of the 132 cultures attempted. Had I successfully cleansed the area I should probably have had no growth in any case.

To eliminate this element of obvious error I devised a method which was apparently successful. The results this led to are described below.

AUTHOR'S TECHNIC

Since the term "infection" presumes invasion of the tissues by bacteria, I next attempted the culture from said tissues of whatever organism might be present in the pruritic tissue. Recognizing the difficulties of this, in view of my previous findings as to the bacteria on the surface of such tissue, I devised the following technic: After thoroughly washing the pruritic area with sterile soap and rinsing with sterile water, under complete aseptic technic and with sterile instruments, I removed a small section of tissue from this area and dropped it into a tube of sterile broth. The cotton plug was flamed and also the tube mouth, and the tube placed in an incubator. Here it was kept at 37 C. for twelve hours, when the tissue was removed by means of sterile forceps, rinsed in sterile water, and dropped into another sterile tube containing 5 per cent. phenol aqueous solution. Here it was allowed to remain for five minutes. Such treatment is considered by bacteriologists as sufficient to kill all living bacteria within reach of the fluid. I then removed this piece of tissue and rinsed it thoroughly in sterile water, and under continued aseptic measures cut it into four pieces, all of which I placed in another tube of sterile broth and incubated as in the first instance, except for a longer time, twenty-four hours being allowed.

The rationale of this technic is as follows: By my first step I attempted to obtain a culture of organisms still lodged on the surface. By exposing the tissue to phenol solution I aimed at destruction of such surface organisms as existed after cultivating; then, working under the assumption that I had destroyed all bacterial life on the exterior of my specimen, I laid its interior open with the object of obtaining a culture from that part of the specimen.

I did this with eleven specimens. My results were as follows: From the first culture I obtained seven growths out of the eleven taken, and six showed *B. coli.* Three were of the communis species and one was *B. coli communior.* In four of the seven there was also a growth of the gram-positive diplococcus. In one also there was a growth of staphylococcus.

In my second culture I obtained only three growths out of the eleven taken. All three showed only one organism, *B. coli.* Only one of three had shown surface growth by first culture.

In checking over these three cases I stumbled on a fact which has since proved of clinical importance to me in the treatment of these cases. All three of these had excoriated areas on examination, whereas the other cases apparently did not. It seemed to me, therefore, that this break in the natural barrier had permitted or made easy the bacterial invasions which my cultures seem to indicate. To check up on this I took one case with and one without excoriations and ran them through

the same process. The results confirmed my suspicions, as *B. coli* was cultured from the former and no organism from the latter. Here, then, I reasoned, we have presumptive evidence of bacterial invasion occurring in cases which clinically present evidence of epidermal injury. To demonstrate this beyond the peradventure of a doubt, and moreover to confirm the nature of such infection, I next attempted to demonstrate the bacteria in situ in the tissues.

To this end I then procured eight specimens in cases of excoriations, and proceeded to attempt demonstration of the bacteria in situ.

As is commonly known, the demonstration of micro-organisms in the tissues is at best a difficult task. Even in tissues or purulent exudate known to contain bacteria, it is surprising to see how few may be found, in any given field. In addition to this difficulty arising from the low numbers to be found in any particular area, there is the further complication that the protoplasm of bacteria responds to staining reagents in a closely similar manner to the nuclear chromatin of the tissue cells. Thus we are burdened with the possibility of an honest error in differentiating nuclei from micro-organisms. Not only nuclei, but also precipitated stain, chromatin granules, dirt on the slide or cover glass and various mitotic figures must be distinguished before we are certain that an object we discover is really organic in nature. In the skin the presence of normal pigment granules is one more fact to be borne in mind. All of these difficulties are experienced when searching for bacteria, particularly gram-positive organisms. When it comes to gram-negative bacteria, the Gram differentiation method is rendered useless because of the fact that the tissue cells themselves (exclusive of the nuclei) also stain gram-negative. As one of the organisms under suspicion is the gram-negative bacillus *B. coli*, the realness of these difficulties becomes especially apparent.

In obtaining the specimen to be subjected to this sort of an investigation great care must be taken, and it should be done with as little handling as possible. The use of mouse tooth forceps is to be avoided, as the specimen is torn by this instrument. A hemostat, on the other hand, crushes so as to render structure imperfect. The ordinary biopsy punch is not very well adapted for use in this region. Best results are obtainable from the use of an Allis clamp or anatomic forceps lightly held. Small pieces are preferable to large ones for the reason that although more difficult to work with, they allow better permeation of the fixing, hardening and staining reagents.

The early fixation of the specimen is desirable so that postmortem changes may be reduced to a minimum. Although many fixatives are available, the most satisfactory in my hands has been Zenker's fluid and 80 per cent. alcohol. The use of absolute alcohol impairs the finer structure of the tissue, which it is of course desirable to preserve in a

study of this kind. Corrosive sublimate has a tendency to make the tissue brittle and to precipitate in minute quantities in the tissues, thus leading to undesirable confusion with micro-organisms. The small amount of this substance in Zenker's fluid does not, however, appear to exert such an action. I have used formaldehyd (from 5 to 10 per cent.) with fair results.

I have followed this method in my studies:

1. Fix in Zenker's fluid for twelve hours.
2. Wash in running water for twenty-four hours.
3. Ninty-five per cent. alcohol for twenty-four hours.
4. Absolute alcohol for twenty-four hours.
5. Acetone for two hours.
6. Xylol until clear.
7. Embed.

Embedding may be done in celloidin or in paraffin. The latter is by far the most preferable for this work, for the reason that celloidin itself takes the stain to a certain degree and makes the distinguishing of bacteria unduly difficult. To embed in paraffin we place the specimen in two successive baths at a temperature of 56 C. for two hours each. The embedded specimen is then mounted on a sectioning block and chilled. Sections are cut on the following day. These are best cut from 3 to 6 microns thicks.

The staining of these sections is most conveniently done with the sections attached to the slides. This may be accomplished by covering the slide with a thin layer of a filtered mixture of equal parts of egg, albumin and glycerin, to which a small crystal of camphor has been added. The section is then floated on a slide so prepared and set away in the thermostat for from four to five hours. After this they may be stained.

As to the stains I used in this series: In an endeavor to demonstrate organisms in the tissue—particularly *B. coli,* which I felt certain I should find—I used more than twenty-two different staining methods or modifications of these. The best results were obtained from Nicolle's modification of Loeffler's method, Hiss and Zinsser's modification of Gram's method and from McCallum's modification of Goodpasture's method. The technic of these methods is as follows:

TECHNIC OF METHODS OF STAINING USED

NICOLLE'S MODIFICATION OF LOEFFLER'S METHOD

1. Alcoholic methylene blue—from five to fifteen minutes, or Loeffler's methylene blue—from one to twenty-four hours.
2. Wash in 1:1,000 acetic acid.
3. Wash in 10 per cent. tannic acid for a few seconds.
4. Pour absolute alcohol over the section for from ten to twenty seconds.

5. Place in xylol till clear.
6. Mount in balsam.

When using this method I have found it advisable to counterstain with eosin.

HISS AND ZINSSER'S MODIFICATION OF GRAM'S METHOD

1. Anilin-water gentian violet for from five to ten minutes.
2. Wash in water.
3. Cover with Gram's iodin solution for one minute.

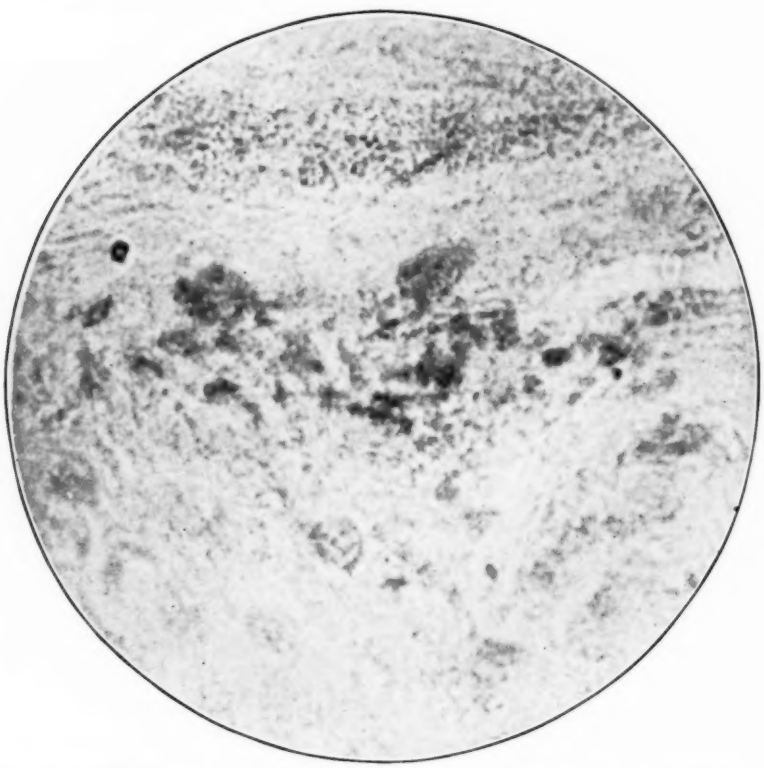

Fig. 3.—Bacteria (staphylococci) in the epidermis of a case of pruritus.

4. Wash in water.
5. Absolute alcohol till no more color leaves.
6. Place in xylol till clear.
7. Balsam.

MC CALLUM'S MODIFICATION OF GOODPASTURE'S METHOD

1. Goodpasture's stain for from ten minutes to one-half hour.

Goodpasture's stain:

30 per cent. alcohol	100 c.c.
Basic fuchsin	0.59 G
Anilin oil	1.00 c.c.
Phenol crystals	1.00 c.c.

2. Wash in water.

3. Differentiate in 40 per cent. (pure) formaldehyd. This requires only a few seconds. The bright red color washes away and gives place to a clear rose color.

4. Wash in water.

5. Counterstain in saturated picric acid. The section should remain until it assumes a purplish color, from three to five minutes.

6. Wash in water.

7. Differentiate in 95 per cent. alcohol. The red color reappears, and some of it is washed out. Some of the yellow of the picric acid is also washed out.

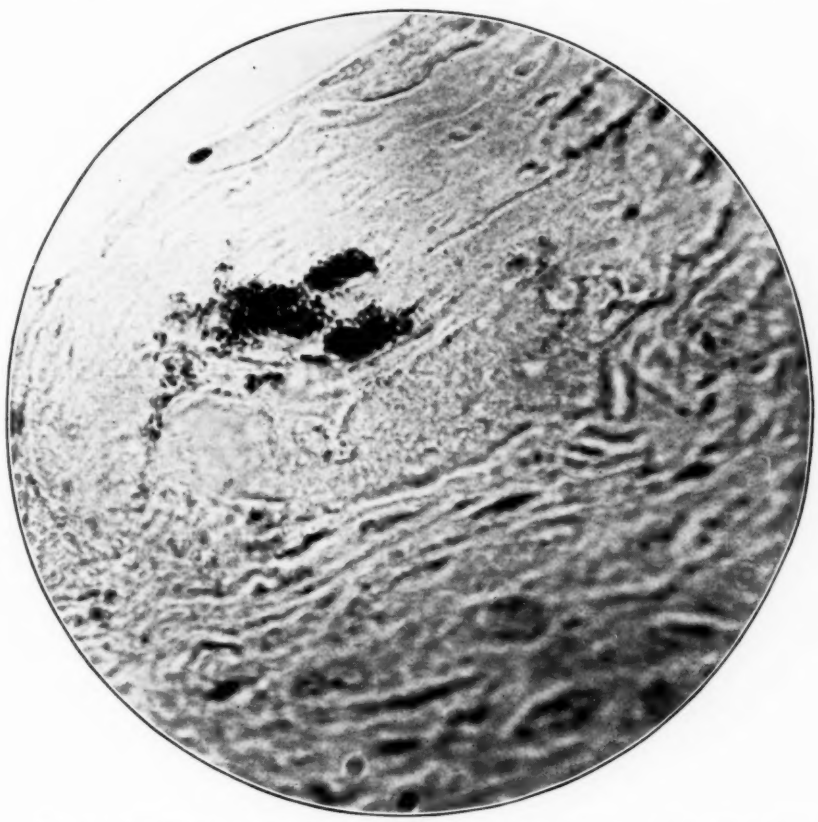

Fig. 4.—Bacteria in the epidermis of a case of pruritus. Note the clumps of staphylococci.

8. Wash in water.

9. Stain in Stirling's gentian violet for five minutes or more.

10. Wash in water.

11. Place in Gram's iodin solution for one minute.

12. Blot dry without washing.

13. Use anilin oil and xylol equal parts until no more of the color comes away.

14. Two changes of xylol.

15. Mount in balsam.

This stains gram-negative organisms red, gram-positive organisms blue and the tissue brilliant shades of red and purple.

Despite the great care in the selection of cases, the obtaining of specimens and the staining thereof, bacteria were found in only seventy-four out of about 1,700 sections examined. This of course must not be construed as being indicative of the absence of bacteria in all but the seventy-four. All that it can logically be assumed to signify is the success of the staining methods used in these instances. The bacteria

Fig. 5.—Staphylococci in the tissues in a case of pruritus. Note the position of the bacteria in relation to the break in the superficial epidermis caused by scratching, or cracking of the skin.

may or may not have been present in the others, but as utmost care and painfully persistent search failed to reveal their presence I feel safe in claiming that they were probably not there. Figures 5, 6 and 7 show sections in which I have been able to demonstrate their presence. They are to be seen in the superficial layers of the epidermis immediately subjacent to a crevice in the surface. Unfortunately, they are stained

light blue, and although perfectly distinct to the eye, they do not photograph well. Under the microscope this specimen shows perfect clumps of staphylococci. In all of the specimens showing organisms present said organism was, morphologically, a staphylococcus, and this was substantiated on culture in each case. In no case did their invasion appear to reach further than halfway to the basement membrane of the epidermis. Likewise in each case there was an evident break in the surface epidermis or a distinct cytolysis of the superficial layers. There was not a single instance in which *B. coli* or *Streptococcus fecalis* was demonstrated in the tissues.

CONCLUSIONS

My conclusion naturally follows that where the epidermis is injured by traumatism or maceration an invasion of bacteria may occur. This invasion in the cases studied is moderate and limited, either due to the resistance of the epidermal cells or to the low virulence of the invading organism.

Since invasion of the tissues has been accomplished by the organism, it fulfils the first postulate of the infection. Its growth in clumps indicates its subsistence there, so that in truth we here have an infection surmised and proved. As previously mentioned, the bacterium found was the staphylococcus. I believe that more cases are due to infection than my findings prove, but I am just as firmly convinced that the percentage is much lower than is commonly supposed.

It is apparent that the causative agent of whatever infective condition is present is not a streptococcus. I consider the occurrence of infection proved, but the evidence favors a different etiology than that of a streptococcus. On the basis of experimental results mentioned previously, I feel certain that both *staphylococci* and *B. coli* can be justly regarded as the probable infective agents. The observations contained in this report suggest a plausible relation between demonstrable infection and visible clinical lesions or alteration in the affected area. It is recommended that this fact be borne in mind in the treatment of cases presenting such lesions. This is particularly the case when vaccine therapy is contemplated. The intelligent use of vaccines will give gratifying results, but their unintelligent application can only result in bringing into disrepute a truly useful therapeutic agent.

540 Park Avenue.

OSSIFYING SARCOMA OF THE SKIN METASTATIC FROM OSSIFYING SARCOMA OF THE HUMERUS

CLARK W. FINNERUD, M.D.

CHICAGO

True bone formation, as well as calcification, has been recorded in a few instances as occurring in the skin at the sites of preexisting cutaneous lesions and trauma, supposedly the result of metaplasia.

Two or three instances are also on record in which bone tumors, single and multiple, developed independently in the skin of young persons, probably as the result of growth of misplaced embryonal cells.

The case herein reported is one of true bone formation in the skin, the result of metastasis of an ossifying sarcoma of the upper end of the right humerus. An exhaustive study of the literature revealed no similar illustration. Cases were reported in which the primary ossifying tumor involved the skin only by direct extension. Sarcomas of the skin of various types, primary and secondary, are not extremely unusual, but apparently there is no other instance in which ossification has been noted, although several cases of giant cell sarcoma of the skin are on record, and in some of these the origin was thought to be the bone marrow.

REPORT OF A CASE

History.—A man, aged 43, when first admitted to Dr. Phemister's service at the Presbyterian Hospital, said that seven months previously he had sprained the right shoulder lifting a trunk, but that he had recovered completely in two days. One month later he noticed that the right shoulder was a little larger than the left, and in about another month there was a continuous aching sensation in the shoulder joint. The swelling gradually increased in size, and the pain increased in severity during the next two months and then remained stationary. About two months before admittance to the hospital, limitation of motion set in.

Physical Examination.—On admittance to the hospital the right shoulder was slightly larger than the left, its bony landmarks were a little less prominent, and the skin covering the right shoulder and upper third of the right arm was slightly red. A bony hard spindle shaped enlargement of the upper end of the humerus could be felt, beginning at about the level of the insertion of the deltoid. Slight limitation of motion was present, so that hyperflexion, extension and abduction were either impossible or accompanied by sharp pain. There were no axillary adenopathy and no definite tenderness. A roentgenogram showed a dense shadow cast by the enlargement about the periphery of the upper end of the humerus.

First Operation.—This was performed the day after the patient entered the hospital. The humerus was disarticulated from the scapula and the upper half of the humerus was resected (Fig. 1). A bone transplantation from the left

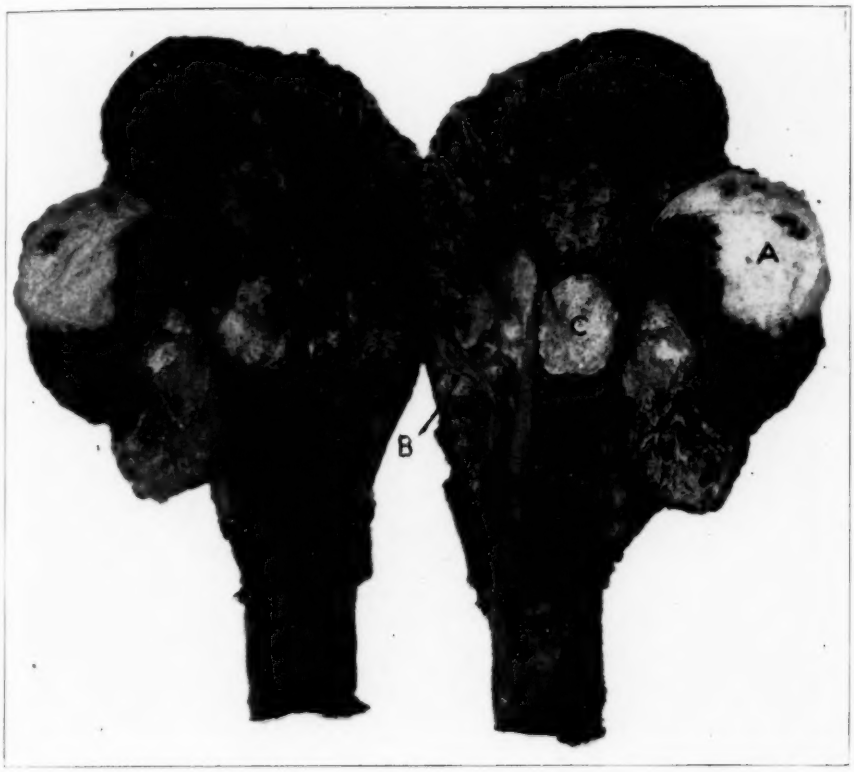

Fig. 1.—Longitudinally sectioned specimen removed at first operation. The light areas are unossified; *A* consists of cartilaginous tumor; *B* and *C* of spindle cell sarcoma.

Fig. 2.—Anteroposterior and lateral roentgenograms of excised ossified cutaneous metastasis in the left submaxillary region.

Fig. 3.—Primary tumor illustrating the spindle cell area in the medullary cavity of the upper end of the humerus. × 130.

Fig. 4.—The cartilaginous portion of the original tumor. × 130.

fibula was performed. The patient was discharged from the hospital five weeks later; reporting at intervals for postoperational roentgen-ray treatments.

Second Admission.—The patient reentered the hospital sixteen months after the first operation with a large bony hard recurrence in the region of the original tumor. It had become extremely painful and had produced marked edema of the extremity during the previous three months. A small lump in the scalp was noticed in the left parietal region about two months before the second admittance to the hospital and another in the skin of the left submaxillary region at about the same time.

Physical Examination.—There was a firm nodule about 1.5 cm. in diameter in the scalp of the left parietal region, which was not adherent to the underlying bone. There was a bony hard nodule of about the shape and size of a small cherry in the skin of the left submaxillary region. The right upper extremity was markedly edematous, and the skin from the elbow to the clavicle and scapula was discolored from radiotherapy. There was a hard mass of the upper and inner surface of the humerus which was continuous with the bone and which extended downward for about 6 inches (15.24 cm.). The shoulder region was tender and painful.

Second Operation.—This was performed the day after admittance, consisting of an intrathoracoscapular amputation for relief of pain. Ten days later the ossified nodule of the skin of the left submaxillary region was excised (Fig. 2), and five weeks later the scalp nodule was removed.

Present Condition.—Nine months after the second operation the patient had ossifying metastases in the lungs and pleura (which are causing great respiratory embarrassment), one bony hard nodule in the scalp of the occipital region and a similar one in the skin near the right angle of the mouth.

Pathologic Examination of the First Specimen.—This showed grossly a variety of types of tumor on longitudinal section of the humerus (Fig. 1). Microscopically, some regions showed spindle cell sarcoma, others chondrosarcoma, and still others ossifying sarcoma (Figs. 3, 4 and 5).

Pathologic Examination of the Second Specimen.—Gross examination revealed the surrounding muscles and even the skin invaded by the ossifying tumor. The transplant, which had been fractured, was almost completely surrounded by the tumor mass. Microscopically, the characteristics were similar to those of the original tumor except that one section from the periphery with but little ossification in it showed numerous large multinuclear giant cells and some degeneration.

Histologic Examination of the Skin Tumor of the Left Submaxillary Region.—Practically the entire metastasis had undergone immature ossification, being composed chiefly of anastomosing irregular bony trabeculae and cancellous spaces occupied by areolar tissue containing some fat and a few marrow cells (Fig. 2). There were no superimposed lamellae or other signs of mature ossification. The ossifying tumor extended from well in the subcutaneous tissue to the epidermis, existing as cancellous bone in orderly disposition throughout the reticular portion of the corium and as osteoid tissue in a connective tissue matrix throughout the papillary corium. In the cancellous spaces blood vessels were fairly numerous and throughout the corium and subcutaneous tissue true bone cells were of frequent occurrence. A thin layer of areolar tissue separated the encapsulating spongy bone from the hair follicles and skin glands. Interlacing swollen and clublike collagen fibers, partially calcified, occupied the uppermost portion of the derma (Figs. 6 and 7).

Fig. 5.—Section from the original tumor, magnified about 65 diameters, composed of almost mature tumor bone with a few areas of spindle cell sarcoma in the inferior portion.

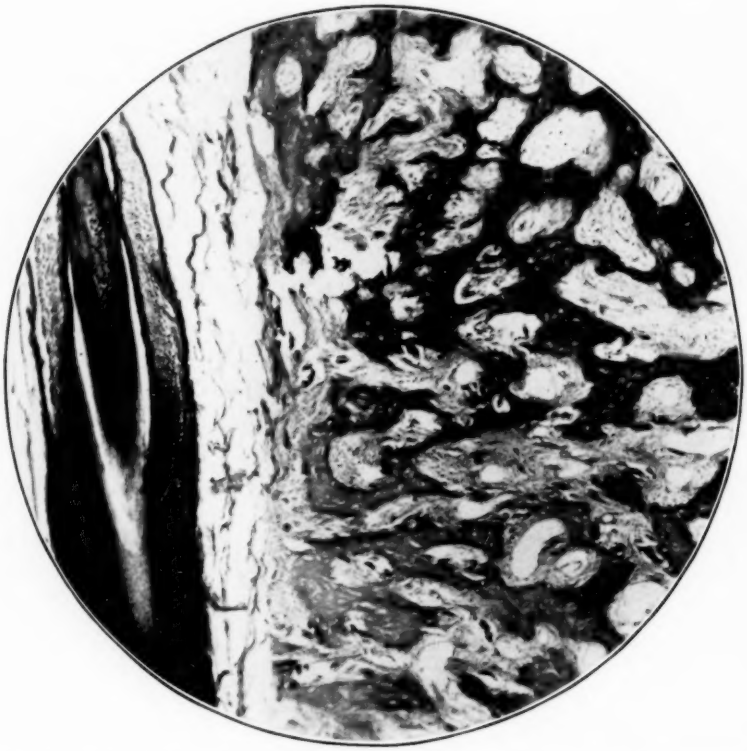

Fig. 6.—Skin metastasis in the left submaxillary region showing cancellous bone extending into papillary portion of corium. × 65.

Fig. 7.—Skin metastasis in left submaxillary region showing immature bone about sebaceous gland and hair follicle. × 60.

Fig. 8.—Section of scalp metastasis showing spindle cell sarcoma; *A*, osteoid tissue about hair follicles and sebaceous glands. × 65.

Histologic Examination of the Scalp Tumor.—There was a spindle cell sarcoma containing considerable osteoid tissue but no real bone (Fig. 8). The spindle cell proliferation was of the same type as that part of the original tumor and occupied approximately and to the same extent that portion of the corium and subcutaneous tissue occupied by cancellous bone in the metastatic lesion of the skin of the left submaxillary region. Osteoid tissue, chiefly in the form of swollen connective tissue fibers with some calcification and a few bone cells, was present and for the most part throughout the upper corium but to some extent in the reticular portion where it was most marked about the hair follicles and sebaceous glands. No cartilage cells were seen in either cutaneous metastasis.

25 East Washington Street.

THE STATUS OF THE COMPLEMENT-FIXATION TEST IN RELATION TO THE CURE OF SYPHILIS

ROBERT A. KILDUFFE, A.M., M.D.
LOS ANGELES

The physician confronting a patient with a diagnosis of syphilis is likely to be confronted, in turn, with several pertinent queries: 1. Is the disease curable? 2. How long a time will be required? 3. What is the amount of treatment necessary to achieve a cure?

An honest and truthful answer to these questions, of importance to physician and patient alike, is not to be made off-hand, because of their relation to matters of moment in regard to the future of the patient: the permissibility of marriage, the possibility of infected or noninfected offspring, and the like.

With regard to the curability of syphilis, it can be definitely stated that the disease is curable, as is proved by the occurrence of reinfections; * but it must be no less definitely understood that, in the absence of such drastic and undesirable method of proof, satisfactory and indisputable criteria are difficult to formulate and equally difficult to demonstrate. In other words, although the disease is curable, he is rash who takes it upon himself to state definitely the moment at which a cure has been achieved.

This phase of the subject has been well summed up by Buzzard: [1]

> If you take a hundred cases of syphilis and treat them all according to the standard methods of the day you are justified in believing that a certain proportion of them will have been cured, but you cannot honestly tell any single one of these patients that this disease has been eradicated. . . I am optimistic enough to believe that if this truthful line were adopted and the patients urged to have periodic courses of treatment for the rest of their lives, we should see very much less of the late syphilitic diseases than we do.

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* While it has been commonly accepted as true heretofore that syphilis cannot be implanted on syphilis and that the occurrence of a second chancre was evidence of the cure of the previous infection, recent investigations by Brown and Pearce cast grave doubt on this assumption.

These investigators (Brown W. H., and Pearce L.: Superinfection in Experimental Syphilis Following the Administration of Subcurative Doses of Arsphenamine and Neorasphenamine, J. Exper. Med. [May] **33, 5**:553, 1921) have shown that superinfection can occur in the rabbit when the animal has been insufficiently treated and it is quite possible, therefore, that in the human being we must bear in mind the necessity for distinguishing between reinfection and superinfection. This fact augments the importance of extreme care in the evaluation of the significance of the negative complement-fixation reaction in its relation to the treatment and cure of syphilis.

1. Buzzard, E. F., in Fraser: Lancet, Jan. 8, 1921, Note 2.

Any one interested or engaged in the treatment of syphilis cannot fail to be impressed with the fact that a large percentage of cases is composed of patients who have been treated in the past, who had been told that they were cured, and who return with serious and often intractable late manifestations; and to any one pondering the situation, it is obvious that this will always be true until a satisfactory standard of cure shall have been achieved.

In the light of our present knowledge—and there is little to suggest that future investigations will be of much avail in remedying the situation—there is *no absolute evidence of cure possible,* and it is necessary to appreciate, therefore, that a standard of cure must be looked on as a relative phrase only; what is aimed at of necessity is the rendering of the disease sufficiently quiescent, latent and innocuous to prevent the occurrence of late sequelae.

This conception of the treatment of syphilis has been well emphasized by numerous authorities, among whom may be noted Fraser,[2] who emphasizes that the standard of cure in syphilis must depend on the stage at which the disease is seen, the type of the disease and the manner of treatment adopted.

In the absence, therefore, of definite and indisputable evidence of the cure of syphilis, in the presence of cumulative and absolute evidence that the disease may remain latent and asymtomatic and yet capable of renewed, active, grave and disastrous manifestations; and that no set number of injections of arsphenamin nor stated amounts of mercurials can be safely relied on as constituting a sufficiency of treatment, it was inevitable, following the introduction of the Wassermann test as a means of detecting the presence of syphilis even in the absence of clinically demonstrable symptoms, that this procedure should gradually become in a more or less definite way the standard by which the cure of the disease was to be determined. This tendency has grown to such proportions that there are no small number of practitioners whose treatment of a given case is more or less primarily focused on the securing of a serologic negative and whose standard of cure is limited and defined by a certain set number of such serologic negative reactions secured during a more or less well defined period of time.

The exact number of negative reactions to be obtained and the exact period during which they shall be secured are, of necessity, somewhat variable quantities; it is, nevertheless, true that in a definite proportion of cases the treatment of syphilis has been transformed into the treatment of the Wassermann reaction—a condition which has been and must be the subject of widening protest in the interests of the patient.

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2. Fraser, A. R.: The Standard of Cure in Syphilis, Am. J. Syphilis **6**:633 (Oct.) 1922.

The subject is not without its inherent difficulties; not the least of which, as has been noted elsewhere,[3] is the unwarranted importance given to the face value of a negative reaction.

Unfortunately for the clarification of a complex subject, the exact mechanism and rationale of the complement-fixation test in syphilis remains, as yet, unknown. The reaction is not biologically specific, nor has it been definitely shown that the substances producing the reaction are of spirochetal origin either directly or indirectly, although there is much to warrant the latter assumption as being at least logical.

Syphilis is, primarily, a tissue disease, the symptoms of which to no small extent represent a defensive tissue reaction; and the theory has been advanced—with much in its favor—that the complement-fixation reaction is an expression of an interaction between the tissues and the spirochetes or their products, or both. This is, at least, a logical assumption and one borne out by much available clinical and experimental evidence. Obviously, then, the treatment of syphilis is, as it were, inextricably associated with the complement-fixation test which —when wisely interpreted—may be a valuable adjunct.

If, as must be admitted, the complement-fixation test is not a biologically specific test, it is, nevertheless, a procedure which has been tried out in hundreds of thousands of cases, in which trial it has withstood numerous assaults, invectives and criticisms. Modern refinements of technic have rendered it incomparably more delicate and reliable than the original procedure, and have achieved for it a high degree of relative specificity; the technic described by Kolmer and his associates,[4] for example, bids fair, as evidenced by the results of over 30,000 tests so far reported, to prove specific for syphilis, with the single exception of yaws. This perfection of technic, while it is an advantage from the standpoint of treatment in that it renders the obtaining of negative reactions more difficult and so leads to more prolonged treatment, is, however, also a possible source of danger unless it is emphasized and continuously realized that the test must always be subjected to interpretation and evaluation.[5]

The proper interpretation of the significance of complement-fixation tests, therefore, is of paramount importance, especially when the results of the test are to be used as a standard by which therapeutic efficiency

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3. Kilduffe, R. A.: The Status of the Negative Wassermann Reaction, J. A. M. A. **79**:2215 (Dec. 30) 1922.

4. Kolmer, J. A.: Studies in the Standardization of the Wassermann Test, Series of Thirty-Two Papers in the Am. J. Syphilis, Beginning **3**:1, 1919.

5. Kilduffe, R. A.: The Clinical Evaluation of the Wassermann Reaction, Arch. Dermat. & Syph. **6**:147 (Aug.) 1922. The Kolmer Complement-Fixation Test for Syphilis: Its Relation to the Clinician and to the Requirement for a Standardized Technic, Am. J. M. Sc., *to be published.*

is to be determined. This must be especially emphasized because as long as absolute evidence or proof of cure is denied, we are forced to rely on the properly interpreted revelation of the complement-fixation reaction.[6] This being the case, it is of interest as well as of practical importance to determine the status of the complement-fixation test in relation to the cure of syphilis and to what extent the reaction may be relied on as an indication of clinical cure and as a guide to the extent of treatment.

TWO INTERPRETATIONS OF THE WASSERMANN TEST

Two different interpretations are probably made of the Wassermann test: one group of physicians relies more or less absolutely on serologic negatives as a justification for the cessation of treatment and the dismissal of a patient; the other, while conceding to the complement-fixation test a valuable place in the diagnosis of syphilis, denies it a place in the treatment of the disease. The former group is composed largely of those whose contact with and handling of the disease is more or less casual.

There can be little doubt as to the significance of a positive reaction when properly checked and controlled: it is an indication of an interaction between the tissues and the spirochetes and hence, of necessity, of the necessity for further treatment. This statement is susceptible of clinical as well as experimental verification regardless of the absence or presence of clinically demonstrable or detectable symptoms. Not infrequently a positive reaction constitutes the only available evidence of the disease, especially when, as emphasized by Fordyce,[6] the lesion responsible for its occurrence is in the cardiovascular system. This observer notes that necropsy shows in asymptomatic syphilis with a positive complement-fixation reaction that the aorta is the site of the lesion in 90 per cent. of cases, while in 40 per cent. there is involvement of the viscera. The same opinion is also expressed by Stokes and Busman,[7] whose study of 101 asymptomatic cases presents convincing evidence that a careful and minute study of such cases is not only necessary, but will also demonstrate especially the presence of pathologic lesions of the cardiovascular system. A significant conclusion as a result of their study was that a persistent positive reaction in the absence of gross manifestations of syphilis was to be looked on as more than a single symptom and rather as a guide to an underlying multiplicity of symptoms.

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6. Fordyce, J. A.: The Prognosis of Syphilis, Am. J. M. Sc. **166**:3 (Sept.) 1923.

7. Stokes, J. H., and Busman, G. J.: A Clinical Study of Wassermann-Fast Syphilis with Special Reference to Prognosis and Treatment, Am. J. M. Sc. **60**:584 (Nov.) 1920.

It is not the positive, but the negative, reaction that is a source of confusion. It is the malinterpretation of the significance of a negative or a series of negative reactions which is, peradventure, responsible for the occurrence of a definite proportion of the late manifestations of the disease.

It is true that the theory has been advanced that syphilis so alters the tissues that a positive reaction may persist even after a cure has been attained, but the bulk of the evidence is against such a supposition, which may be looked on as untenable.

Refinements of technic have rendered the obtaining of a negative reaction more difficult, but refinements of technic will never alter the fact that it is not the infection but the tissue reaction to infection which produces reagin; in the presence of latent or quiescent infection not productive of such interaction and hence of reagin in detectable amounts, the reaction will be negative, but the infection will still be present although perhaps asymptomatic.

In the light of our present knowledge, therefore, it will never be safe to establish an absolute minimum or maximum of serologic negatives as indisputable evidence of the cure of syphilis. The treatment of syphilis should be the treatment of the person who has the disease, and the amount and duration of his treatment will depend on the stage and type of the disease when first seen.

Shall the treatment of the disease be directed toward the attainment of negative serologic results? The weight of both laboratory and clinical evidence forces an affirmative answer, and the careful clinical and laboratory study of "Wassermann-fast" cases reinforces it.

Can any definite number of serologic negatives give positive assurance of cure? This query must be answered in the negative, and any tendency toward the laying down of a specific number of negative reactions must be strongly decried. Serologic negative reactions *alone* are insufficient evidence of probable cure, and the assumption that a certain number of negative reactions indicates a cure is unfounded and an injustice to the patient, and an undue reliance on such proof alone renders him liable to late and grave manifestations of the disease.

What, then, can be said of the value of a negative reaction? Simply that it is an evidence of a lack of interaction between the body cells and the infecting organisms. With each subsequent negative reaction the sense of protection of the patient against later assaults of the disease becomes more marked, but no cumulative number of negative reactions will ever suffice for the hazarding of more than a statement of probabilities—such a patient is *probably* cured.

Under these circumstances, has the complement-fixation test any place or value in the treatment of syphilis? While there are those

inclined to answer this query in the negative, this attitude may be disputed.

One of the most important and essential features of the treatment of syphilis lies in the persistence of the patient in his treatment. All patients are amenable to any remedial measures while there is visual or subjective evidence of the disease on the principle that: "The devil was sick—the devil a monk would be." With the disappearance of symptoms, however, this avidity of the patient for further treatment undergoes a marked recession: "The devil got well—the devil a monk was he."

The persistence of a positive reaction in the absence of subjective symptoms in such cases is a potent factor in convincing him that further treatment is needed, and the subsequent gradual decrease, especially when the test is quantitatively made, an encouragement to still further perseverance.[8]

Moreover, if both the positive and the negative reactions are placed before the patient in their true light, he will be cognizant of the fact that a negative reaction is merely a statement of the serologic situation at the moment of testing, and appreciative of its lack of standing as a guarantee of the perpetuation of such a serologic condition. He will also be more appreciative of the necessity, not of securing merely a stated number of such negative reactions, but of obtaining these as *an incident in the course of a search for a recurring positive reaction.*

If the practitioner also is equally cognizant and appreciative of the same important facts, the relation of the complement-fixation test to the treatment of syphilis will rest on a more rational basis than it does at present in some quarters.

It would undoubtedly be wise and for the ultimate benefit of the patient if all were taught at the onset of the infection that continual vigilance is the price of safety and that continual observation is a sine qua non of the treatment of syphilis; also that periodical complement-fixation tests and, perhaps, periodical courses of treatment every year or so, regardless of serologic results, are advisable. If such a regimen were rigorously adhered to, the incidence of late manifestations of the disease would undoubtedly decrease.

It is greatly to be regretted that the complement-fixation reaction has been relied on both in diagnosis and treatment to the detriment of the clinical study of syphilis; and only when it is recognized in its true light as a *phase* of the disease, as a single and at times isolated symptom, to be interpreted in conjunction with all the other symptoms, will the best possible results be obtained.

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8. Kilduffe, R. A.: A Note upon a Graphic Method of Reporting the Wassermann Reaction, J. Lab. & Clin. Med. **9**:1 (Oct.) 1923.

Correspondence

SIR MALCOLM MORRIS MEMORIAL FUND

To the Editor:—The death of Sir Malcolm Morris, K.C.V.O., F.R.C.S., Ed., on February 19, marks the passing of one who was widely known and greatly respected, both by his professional colleagues and the public generally, for his work in connection with dermatology, tuberculosis, public health and medical literature.

To perpetuate his memory it was resolved at a recent meeting of representatives of the various hospitals and organizations with which he was particularly associated, to endeavor to raise a sum of money sufficient to provide for an annual lecture on the preventive aspects of public health and dermatology. If funds permit, it is hoped also to endow a bed for patients with skin disease at St. Mary's Hospital, London. These objects are known to accord with Sir Malcolm Morris' expressed view that the only fitting memorial to a life of successful endeavor is one that carries on the causes to which that life was devoted.

A representative Committee has been formed, and we invite contributions, in any amounts, which will be gratefully acknowledged if forwarded to the Honorary Treasurers, "Malcolm Morris Memorial Fund," at 12 Stratford Place, London, W.1.

THOMAS HORDER,
Federation of Medical and Allied Services.
E. B. TURNER,
British Medical Association.
E. GRAHAM LITTLE,
St. Mary's Hospital.
C. NEVILLE ROLFE,
Nat. Council for Combating Venereal Diseases.
W. GRIFFITH,
St. John's Hospital.
A. E. HAYWARD PINCH,
Radium Institute.
H. T. HERRING,
Cremation Society of England.
N. HOWARD MUMMERY,
Honorary Secretary.

"GLOSSITIS RHOMBICA MEDIANA"

To the Editor:—In Dr. J. E. Lane's paper on "Glossitis Rhombica Mediana," which appeared in the May issue, the author states that "Nothing has appeared in English on the subject." The same disorder was described by Drs. Fordyce and Cannon in the December, 1923, number of the ARCHIVES as "A Hitherto Undescribed Condition of the Tongue."

It may be of interest to note that this disease, first published in 1914 by Brocq and Pautrier, is briefly but adequately described in English on page 226 of the American translation of Darier's "Text-book of Dermatology," Philadelphia, Lea and Febiger, 1920, under the name of "Median Rhomboidal Glossitis."

S. POLLITZER, M.D., New York.

Abstracts from Current Literature

THE RELATIONSHIP OF NEURODERMITE TO ALOPECIA. G. NOBL, Dermat. Wchnschr. **77**:1320 (Nov. 10) 1923.

In the opinion of the author, both conditions are due to the same trophoneurosis.

URTICARIAL (ANAPHYLACTIC?) DRUG ERUPTION FOLLOWING THE TREATMENT OF PSORIASIS. JOSEF ZOLLSCHAN, Dermat. Wchnschr. **77**:1323 (Nov. 10) 1923.

A 10 per cent. cignolin ointment was applied to a stubborn psoriatic eruption until albuminuria forced its discontinuance. The psoriasis disappeared, and four days later a pruritic urticarial eruption appeared *around* the sites of the former eruption, a possible argument in support of the parasitic theory.

TREATMENT OF PSORIASIS VULGARIS WITH INTRAVENOUS INJECTIONS OF SODIUM SALICYLATE AND THE APPLICATION OF OINTMENTS. ALFRED HOLLANDER, Dermat. Wchnschr. **77**:1325 (Nov. 10) 1923.

In several cases the author injected from 10 to 20 c.c. of a 20 per cent. solution twice a week eight or ten times, simultaneously using an ointment containing salicylic acid and ammoniated mercury for the face and scalp, and salicylic acid and cignolin in an ointment for application elsewhere. In five cases the lesions disappeared within from three to four weeks, while four others did not respond so well. The treatment apparently delayed recurrences.

SUBACUTE NONGENERALIZED DERMATITIS EXFOLIATIVA. EMANUEL WOHLSTEIN, Dermat. Wchnschr. **77**:1327 (Nov. 10) 1923.

A man, aged 58, previously in good health, presented an exfoliative dermatitis of the face, scalp, hands, neck, shoulders and sacrum, of five months' duration, which had started with an inflammation of the right hand, accompanied by chills and fever. Complete recovery ensued within four months. This is considered to be a case of abortive primary dermatitis exfoliativa.

A CASE OF SPOROTRICHOSIS OF THE GENITAL AND ANAL REGIONS AND OF THE ORAL MUCOSA. MENAHEM HODARA, Dermat. Wchnschr. **77**:1328 (Nov. 10) 1923.

In a woman, aged 50, who had had the condition for a year, the lips, tongue and buccal mucosa were stippled with erythematous, ulcerated and grayish membranous patches, while in the genital and perianal region were nodules, pustules and verrucous plaques. Material from the nodules was put on Sabouraud's medium and a pure culture of sporothrix was obtained. Four other cases are alluded to, including one with oral lesions.

CARCINOMA OF THE RECTUM AND SYPHILIS IN A YOUNG PERSON. EDUARD WELLMANN, Dermat. Wchnschr. **77**:1330 (Nov. 10) 1923.

A woman, aged 23, who had received antisyphilitic treatment for six months, was found to have an extensive carcinoma of the rectum, which metastasized to the liver, with a fatal outcome.

A Peculiar Lupus Erythematosus in Combination with Erythema Induratum and Papulonecrotic Tuberculid (Lupus Erythematosus Discoides et Acneiformis, with a Herpetiform Recurrence). G. Nobl, Dermat. Wchnschr. **77**:1373 (Nov. 24) 1923.

In a woman, aged 24, who had had erythema induratum of the legs for one and one-half years, there was disseminated lupus erythematosus of the eyelids, cheeks and ears, of two months' duration, and papulonecrotic tuberculids of the soles and dorsa of the feet. Histologic study of apparently typical acne nodules on the chest and back revealed a picture suggesting that of lupus erythematosus. An outbreak resembling erythema multiforme, involving the face and neck, occurred while the patient was under observation.

Parkhurst, Toledo, Ohio.

A Clinical and Experimental Study of Psoriasis. Karel Gawalowski, Ceska Dermat. **5**:1, 1923.

The author discusses the relationship of psoriasis to hypofunction of the thymus. The results of the author's study collaborate the works of Samberger and Brock, particularly their observation that by increasing the function of the thymus—by means of small doses of roentgen rays to the thymus region—it is possible to decrease or even to remove the parakeratotic diathesis. An extensive study of the blood changes following irradiation of the thymus showed clearly that the parakeratotic diathesis is not directly influenced by end-products of destroyed blood cells. These products may have an indirect effect on psoriasis in some cases by increasing the defensive forces of the organism through resulting inflammatory changes. The work also proves experimentally that there is a slight rise in the pigment formation as a result of an increased thymus function.

The first part of the work contains a review of literature on the subject, a study of morphology, physiology and pathology of the thymus, and the nature and origin of its hormone.

Finding of Plaut-Vincent Bacteria in Ulcerated Interdigital Condyloma. Pavel Bentl, Ceska Dermat. **5**:69, 1923.

A girl, aged 18, with leukoderma and numerous condylomas on the external genitals presented ulcerated condylomatous lesions between the toes of both feet. The lesions were covered with a necrotic, grayish, foul smeeling membrane in which were found besides *Spirochacta pallida*, numerous fusiform bacilli and spirilla, a few streptococci and pseudodiptheric rods. The patient was put on bismuth therapy. While all genital lesions disappeared in fifteen days, the interdigital lesions showed no change except a flattening of the condylomas. The necrotic membranes were not affected. They healed completely after two injections of arsphenamin.

This case shows the efficacy of arsphenamin and the inefficacy of bismuth in combating the Plaut-Vincent flora.

Bismuth Therapy. Anton Neumann, Ceska Dermat. **5**:71, 1923.

Bismuth emetin in oil suspension was used in thirty cases of syphilis, with prompt specific results. The spirochetes disappeared after the first injection

in from one to four days. The Wassermann reaction became negative in the majority of primary and secondary cases after one injection. Intramuscular administration is quite painless and causes no indurations. Only one patient developed a gingivitis. In several cases a light grayish discloration along the incisors was noticed. Blood examination showed a considerable decrease of erythrocytes with an increase in lymphocytes and eosinophils a few hours after injection.

THE COMPLEMENT-FIXATION TESTS IN TUBERCULOSIS. KAREL HUBSCHMANN, Ceska Dermat. **5**:80, 1923.

After an extensive study of the literature and on the basis of painstaking serologic experiments and clinical observations, the author arrived at the following conclusions:

The reactions carried on with Besredka's antigen (a culture of Koch's bacilli in egg yolk and distilled water) are specific for the majority of tuberculous conditions.

A positive reaction—except in persons with a positive Wassermann reaction—may be regarded with all probability as a sign of an existent tuberculous infection of a reactive character strong and active enough to stir up the defensive forces of the organism. This reactive power can be increased by certain means. A negative Besredka reaction does not exclude the existence of a tuberculous focus.

Very weak reactions must be interpreted with care and after due consideration of all facts. Weak reactions were found in cases considered clinically as inactive; they were also found in normal persons living in close contact with tuberculous persons. The intensity of the reaction fluctuates in the same person according to the changes in the general condition and in the process.

The interpretation of reactions in skin cases is rendered more difficult by the frequency of pulmonary complications.

The development of reagins in lupus vulgaris does not seem strong, a fact well in accord with the chronic character of the disease. Extensive, progressive types of the condition give a strongly positive reaction.

In order to test the local or cellular immunity, a series of tests was made using the content of vesicles (produced artificially by carbon dioxid snow) instead of serum. The results were practically analogous. In cases of lupus the local development of reagins does not seem to be considerable.

The results of reactions in cases of lupus erythematodes were not uniform enough to point toward a definite tuberculous origin.

Besredka's antigen deteriorates with age, a fact not formally recognized. In making the tests more than one antigen should be used to obtain the broadest possible antigen basis. "Tuberculose-Hautimpfstoff" (antigen H for short) was used in parallel series with Besredka's antigen and gave the same or quantitatively and often qualitatively better results. Antigen H is more specific in patients with a positive Wassermann reaction. Its cloudiness and yellow tint make the readings more difficult. Wassermann's tetralinlecithin antigen seems even superior to antigen H.

Complement-fixation tests have a certain prognostic value. Their diagnostic value cannot be doubted.

SPINKA, St. Louis.

DERMATOLOGIC ABSTRACTS

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

TWO CASES WITH UNUSUAL CALCAREOUS DEPOSITS. F. F. TISDALL and I. H. ERB, Am. J. Dis. Child. **27**:28 (Jan.) 1924.

In a case reported by Tisdall and Erb calcareous deposits occurred in the epiphyses of most of the long bones. The patient lived only a few weeks, and had a peculiar deformity of the jaw and limitation of movement in other joints of the body. The calcium deposits in another case were in the skin and subcutaneous tissues. The underlying condition appeared to be either scleroderma or zeroderma pigmentosum. Blood serum calcium and inorganic phosphorus determinations in both cases were essentially within normal limits.

TREATMENT OF EARLY HEREDITARY SYPHILIS WITH INTRAMUSCULAR INJECTIONS OF SULPHARSPHENAMIN. F. H. BOONE and A. A. WEECH, Am. J. Dis. Child. **27**:39 (Jan.) 1924.

Twenty-one patients under 1 year of age were treated by Boone and Weech with sulpharsphenamin. Apparently, sulpharsphenamin can be given safely to infants in a dose as large as 20 mg. per kilogram of body weight. The reactions to treatment, either general or local, are very slight. In 55 per cent. of the patients, the Wassermann reaction became negative after six treatments. The symptoms of hereditary syphilis rapidly disappear, and the patient's general condition quickly improves. The ease with which sulpharsphenamin can be administered makes it very useful to the practitioner who is not adept at intravenous therapy.

TREATMENT OF LEPROSY. T. B. HEGGS, Brit. M. J. **2**:1253 (Dec. 29) 1923.

Investigation has led to the belief that healthy human beings living in a healthy climate and sanitary surroundings do not develop leprosy, even when inoculated with it. This conviction has been strenghtened by several cases which Muir has seen. The habits of lepers and of those who come in contact with lepers are largely responsible for transmission, especially in house and bed infections; and a hot, moist climate, by leading to scanty protection of the skin by clothing and to irritation of the skin by insects and skin diseases, promotes auto-inoculation. The degree of body resistance is most important and tends to be higher in temperate, healthy climates and in natural, sanitary surroundings. It is lowered by concurrent or intercurrent diseases, constipation, unsuitable diet, pregnancy and many other causes, and it is commonly in connection with one of these predisposing or exciting causes that leprosy first shows itself. Improvement in sanitation, the introduction of dairy farms and market gardens and the development of individualism stamped leprosy out of England.

SYPHILIS AS ETIOLOGIC FACTOR IN MONGOLIAN IDIOCY. D. O. RIDDEL and R. M. STEWART, J. Neurol. & Psychopath. **4**:221 (Nov.) 1923.

Investigations made by Riddel and Stewart have failed to substantiate the contention of Stevens that mongolian idiocy is a form of congenital syphilis, but they seem to confirm his conclusion that the cerebrospinal fluid of the

Mongol has the property of precipitating solutions of colloidal gold in the syphilitic zone. At this juncture the authors consider it unwise to conclude that syphilis plays no part in the origin of this disease.

UNUSUAL CASE OF VITILIGO IN NATIVE OF SUDAN. R. G. ARCHIBALD, J. Trop. Med. **26**:361 (Dec. 15) 1923.

In the comparatively short period of three years an extensive and, in areas, complete depigmentation of the skin of Archibald's patient occurred, and also pigmentation of portions of the hair of the scalp, axillae and pubis. The depigmentation was unassociated with constitutional disturbance, and laboratory investigations failed to elucidate the exciting cause. It was associated with histopathologic changes which consisted, in the main, of atrophic changes of the cells of the stratum granulosum and rete mucosum, together with a diminution or entire loss of pigment in the usual pigment bearing areas of the skin.

CAUSE OF BALDNESS. R. O. STEIN, Wien. klin. Wchnschr. **37**:6 (Jan. 3) 1924.

Stein believes that the theory that seborrhea is the main cause of baldness, is entirely out of the question. Seborrhea affects both sexes, and it also affects the whole scalp of the man. Yet baldness occurs practically only in men, and does not affect the whole scalp. Examining the typical baldness in young men, he had impression that the whole affection is only the pushing up and back of the normal anterior margin of the hair. This led him to observations on the anterior borders of the hair in normal subjects. He found that this border forms an arc in children and women. After puberty, however, the hair at the side of the forehead in man begins to fall out, and he considers this as a typical secondary sex character, which he calls the "frontal calvities of adolescents." It is known that this part of the frontal region is also a point of predilection for secondary sex characters in certain male mammals. Another center of loss of hair is the parietal region. He found the bald triangles on the forehead in a case of premature puberty, and in some women with other masculine characters, especially hypertrichosis. The anterior border of the hair forms a perfect arc in eunuchoids, just as in children. Thus baldness is only a secondary male sexual character on a seborrheic soil.

SYPHILIS AND TUBERCULOSIS. J. W. SAMSON, Ztschr. f. Tuberk. **39**:161 (Dec.) 1923.

Samson examined eighteen syphilitics with tuberculosis and nine tuberculous patients who had acquired syphilis. It seems that this association has not much influence on the prognosis of tuberculosis.

THE CAMPAIGN AGAINST FRAMBESIA IN THE DUTCH EAST INDIES. C. W. F. WINCKEL, Mededeel. v. d. Burg. Geneesk. Dienst, English, Java, 1923.

Winckel states that the systematic control of endemic yaws was started in Java late in 1919, and to date there are official records of 890,000 injected with neo-arsphenamin, while many others have been injected without official registering. In certain regions the disease is a regular scourge, not only disfiguring and mutilating but incapacitating for work, especially when the lesions are on the feet. Winckel regards it as an essential part of the propaganda, without which lasting success is not possible, the defraying of the expense by

the people themselves. This has three main advantages: firstly, the educational, teaching the people that the government does not give away everything for nothing but may reasonably expect something in return when the people are more or less well to do. Neo-arsphenamin in yaws is peculiarly adapted for teaching this, as its effects are so prompt and so striking that wide circles of the populace are impressed. Another advantage is that the campaign undertaken and progressively increasing has been practically no expense to the government. The contributions of the population amount already to several hundreds of thousands of guilders; only in a few districts the owners of the sugar factories or plantations paid for the indigent part of the population. A third advantage is that by asking payment of those who can afford to pay, the number applying for treatment has multiplied enormously. The experience has constantly been that when after some propaganda, the payment system was introduced, the crows flocking for treatment to the injection centers became ten times more numerous. Many who cannot walk are brought in sedan chairs or baskets by their friends. The dose for an adult native is never over 0.45 gm. and the injection is made in the arm almost always. With two skilled male nurse assistants and civil service lay assistants to hold the arm and attend to the payment, 600 or 800 intravenous injections can be made in a day. The doctor's chief difficulty is to exclude scabies, ringworm, leprosy and similar affections among the applicants. An injection is sometimes made when the lesion is differentiated as tropical ulcer, as surprising improvement is often noted. A further advantage of this yaws campaign is that it has helped to educate the 45,000,000 natives of the Dutch East Indies to respect medical science and medical officials. The single injection of neo-arsphenamin does not cure the frambesia, but it abolishes the sources of infection so the disease will grow less prevalent in time. In the village of Pangdeglang it has been actually exterminated. The article is illustrated.

Society Transactions

NEW YORK DERMATOLOGICAL SOCIETY

Regular Meeting, Jan. 22, 1924

WALTER J. HIGHMAN, M.D., *President*

URTICARIA PIGMENTOSA. Presented by DR. FRASER.

A girl, aged 14 months, presented lesions which comprised oval shaped nodules and pigmented macules; these appeared when the child was two and a half months old, and had persisted. The specimen examined responded to the specific reaction for mast cells.

DISCUSSION

DR. WISE said he agreed with the diagnosis, and commented on the small number of lesions.

DR. HOWARD FOX said he thought the case was unusual, not only on account of the scarcity of the lesions, but also because of the reddish color. He agreed with the diagnosis.

A CASE FOR DIAGNOSIS. LUPUS ERYTHEMATOSUS? Presented by DR. LANE.

A. R., a man, aged 24, an Italian, born in this country, said that the condition started as a red spot on the right cheek about two years before, and on the left about a year before. The entire face and forehead and neck above the collar back as far as the ears was red, slightly scaly, and the skin was slightly thickened. At the edges of the lesions on the neck and on the lobes of the ears, the lesions were a brighter red than on the rest of its surface, and there were minute acutely inflamed elevations in these places. The only diagnosis that seemed at all plausible was lupus erythematosus, but there were none of the characteristic plugs which are usually found in a case of such long standing.

DISCUSSION

The diagnosis of lupus erythematosus was accepted by Drs. Kingsbury, Williams, Whitehouse, Schwartz and Wise, the latter considering it so by exclusion.

DR. HOWARD FOX said he thought it was undoubtedly a case of lupus erythematosus. Of the cases of this condition shown during the evening, one was of the "fixed" and another of a more superficial type. This case bordered closely on the type of lupus erythematosus disseminatus. It was quite possible that in this instance the process would later extend to the chest and arms, the usual locations for this form of the disease.

DR. CLARK said he had been inclined to consider the case one of seborrheic dermatitis; the patient had seborrhea of the scalp and scaly patches on the ear, and had lesions like a follicular seborrhea scattered over the outer portions of the face. It did not show a crusted condition with projecting points. Probably if the patient were treated with bland applications the areas of inflammation would subside and the seborrhea could be cleaned up.

Dr. Howard Fox said that not only was the diagnosis made by exclusion, but also from the effect of roentgen-ray treatment. This would tend to rule out an inflammatory condition such as seborrheic dermatitis, which would probably have improved or disappeared under irradiation, while there would have been no appreciable effect on a lupus erythematosus.

Dr. Lane said the patient had been treated for a year or longer along the lines suggested by Dr. Clark, with no improvement. At a distance, the appearance was somewhat that of eczema rubrum with thickening of the skin, but there had never been any oozing or itching. Within the last two or three weeks some of the acute red lesions at the edge had developed, which rather more inclined him to the diagnosis of lupus erythematosus. He had never seen so extensive a case on the face.

Lupus Erythematosus. Presented by Dr. Williams.

F. M., a man, aged 27, an American, a chauffeur, had had the present eruption on his face for two years. This patient had previously been presented at the Academy Section on January 2, because of the interesting condition of his upper lip.

DISCUSSION

Dr. Whitehouse said he agreed with the diagnosis. The lesions on the lip and on the forehead were rather typical.

Dr. Schwartz, Dr. Wise and Dr. Clark said that they agreed with the diagnosis.

Dr. Lane said he was very much interested in the case because of the differences and the similarities to his own case. On the patient that he himself had shown, there were no signs of seborrheic eczema on the back and chest.

Dermatitis Medicamentosa from Phenolphthalein. Presented by Dr. Williams. Previously presented before the Society, November, 1923.

A boy, aged 11 years and 6 months, was first seen in October, 1923, at which time he had a generalized eruption of one week's duration. He stated that during the past two years he had been taking Ex-Lax at irregular intervals, and that following its use the eruption had been aggravated. On Dec. 10, 1923, the laboratory found *Microsporon furfur* on some of the lesions of the trunk.

DISCUSSION

Dr. Bechet said that the condition impressed him as being tinea versicolor. He could not recall ever having seen a case of phenolphthalein rash with such a widespread eruption as in the case under consideration. The rounded, brownish-reddish lesions of a phenolphthalein rash were absent. Without the history, it would be difficult to make the diagnosis of a drug rash.

Dr. Howard Fox said that it was very difficult to make a diagnosis in a case like this. He agreed with Dr. Bechet that most cases of phenolphthalein eruption ran more or less true to form, and this did not look like any case that he had ever seen. The boy had previously had some patches of tinea versicolor, for which he had been treated, but it was doubtful that the treatment had been sufficiently thorough. In asking the patient about bathing, he said he took a bath once a week. It would be interesting to see him after vigorous treatment—daily bathing followed by antiparasitic treatment.

Dr. Williams said the boy was presented especially because he did not look as though he had a case of ordinary Ex-Lax eruption, particularly on account of the disseminated character. As primarily shown, the patches were not that color at all, and every one accepted the diagnosis of an erythema. The patient had patches of tinea versicolor on the waist, but the erythema patches were quite distinct, and they had been steadily fading since he stopped taking the Ex-Lax. The case was presented because it was a peculiar type.

Dr. Bechet inquired as to the possibility of the occurrence of some impure phenolphthalein, or extraneous article in the hundreds of examples of the drug in candy form, as the cause of the drug rash. The source of the rash when named seemed to be Ex-Lax or some other patented candy form of this popular laxative. On the other hand, he had noted that many of his friends and patients had taken phenolphthalein for months and years without developing a dermatitis medicamentosa. It would probably be nearer the truth to ascribe the incidence of the eruption to the popularity of the drug. After taking thousands of doses, some patients would be bound to develop a drug rash.

Dr. Wise said that one of the Vanderbilt Clinic patients with phenolphthalein eruption, when given chemically pure phenolphthalein, would show a distinct flare-up of the cutaneous and mucous membrane lesions.

Dr. Lane said that most patients take one of the proprietary drugs, and not the pure phenolphthalein. That would be a sufficient reason for the greater frequency of these eruptions after them than after the pure phenolphthalein.

Partial Depigmentation of the Face of a Negro. Presented by Dr. Howard Fox.

J. H., a full blooded colored boy, aged 14, first noticed an eruption on the face six months before. It now consisted of irregular shaped patches occupying about half of the area of each cheek. They were partially depigmented, smooth in places and in others showing a fine scaling. There was no evidence of scratching. The boy was born in North Carolina, and had been in New York for the past seven years. No member of his family was suffering from a similar condition.

DISCUSSION

Dr. Wise said that he had great doubts about the parasitic etiology of the condition as a whole, and as Dr. Foulks had said during the evening Dr. Sabouraud regarded the lesion as an impetigo furfuracea. The lesions of caraate or pinta, of which the speaker had seen hundreds of cases on the north coast of South America, were said to be due to an easily demonstrable aspergillus fungus, which provoked a true leukoderma in the affected areas. In the case presented there was nothing but a faint depigmentation.

Dr. Williams said that in making many examinations for fungi on the hands and feet, aspergillus was found, and they were all discredited because they were so common. He could not give any figures showing the percentage. Unless it could be shown that a definite type of case shows aspergillus in more than 7 or 8 per cent., it seemed doubtful whether the aspergillus had anything to do with it. We seem to have gone too far in ascribing diseases to casual organisms which happen to be found on the skin.

Dr. Howard Fox said he agreed with what had been said by Dr. Wise and Dr. Williams—that the parasitic nature of this disease had not been proved by Drs. Pardo and Dominguez. Their work was, however, creditable and

interesting. He did not think the aspergillus they had found in a small percentage of their cases could as yet be regarded as the causative agent. This patient was similar to many he had seen and to the family of three children he had presented at a former meeting of this society. In most of the cases, the greater part or all of the eruption was situated on the face. In some, there was a slight amount of fine scaling.

SYPHILIS. Presented by DR. WISE for DR. FORDYCE.

L. C., a negro woman, aged 24, married, showed lesions on the legs which had been present for five weeks. A tentative diagnosis of erythema induratum of Bazin had been made, but since then a Wassermann reaction had been four plus. The case was presented with the idea of showing the difficulty of making a differential diagnosis between the two conditions at the first glance.

DISCUSSION

DR. BECHET said he did not think from the appearance of the lesions that they were sufficiently indurated to be erythema induratum; they did not resemble the large hard nodular chronic inflammatory lesions so frequently observed in that disease.

DR. LANE said he agreed with Dr. Wise. From the history and progress, there could be little doubt that the lesions were syphilitic. On the other hand, the improvement of lesions of erythema induratum after arsphenamin was often quite prompt.

DR. WHITEHOUSE said he thought it was a case of syphilis.

DR. WILLIAMS said it was impossible to make a diagnosis from the appearance of the condition.

DR. HIGHMAN said he did not agree that arsphenamin caused a disappearance of nonsyphilitic lesions.

GUMMA OF FOREARM (SIMULATING SARCOID OF BOECK). Presented by DR. WISE for DR. FORDYCE.

Mrs. E. T., aged 45, had had the disease for one and one-half years. At the clinic they hesitated between the diagnoses of syphilis and sarcoid. The lesions resembled the latter, but the condition responded to antisyphilitic treatment, and the Wassermann report was four plus.

A CASE FOR DIAGNOSIS: LESION OF THE BACK OF THE NECK. Presented by DR. WILLIAMS.

E. R., aged 23, an American, a clerk, presented an eruption of four years' duration, which at times appeared more prominent, but never entirely disappeared during this period. The Wassermann reaction was negative. A biopsy was made, but no diagnosis possible. The epidermis appeared normal; in the horny layers were isolated elevations of desquamated cells which were surrounded by an inflammatory exudate.

DISCUSSION

DR. LANE said that he did not know what the condition was. It could not be lichen planus with the histologic report as given.

Dr. Wise said he did not know what it was, but he did not think it was either lichen planus or neurodermite. He suggested the possibility of circumscribed leukemia cutis.

Dr. Howard Fox said that the eruption suggested lichen planus, but that had been ruled out by the histologic examination. While it was not possible to make a positive diagnosis, he suggested the possibility of an unusual type of lichenification.

Dr. Williams said he did not think it was an inflammatory condition, for there were areas of distinct atrophy, and he had never heard of lichenification producing such lesions with atrophic centers.

Dr. Highman said he could not imagine that the histologic report gave a clear picture of what was shown histologically. Certainly the papules suggested lichen planus clinically, but the microscopic description indicated nothing of the sort.

A Case for Diagnosis: Tuberculosis Cutis? Simulating a Hare Lip. Presented by Dr. Bechet.

T. M., aged 35, born in Italy, said that the lesion had begun two and a half years previously, and that there had been a gradual loss of tissue since. At the age of 11, he had had a severe bone disease in the right leg, for which he had undergone an extensive operation. At the time of the examination, he showed the usual large linear scar so frequently observed after an operation for bone necrosis. His wife seemed healthy, and was the mother of two healthy children. She had had one miscarriage. On his upper lip, extending from the right nostril, was a triangular void with its base downward, from which the tissue had completely disappeared, giving the typical hare lip appearance. The borders of the triangle were somewhat indurated, and at its upper edge were undergoing an indolent ulceration. This ulcerating part of the lesion was crateriform in character, and suggestive of tuberculosis; yet syphilis or epithelioma could not be excluded. A Wassermann reaction was negative. Despite this negative report, he was given six injections of arsphenamin intravenously and a few salicylate of mercury injections intramuscularly. This treatment had no effect on the lesion, and a Wassermann test made at the termination of the treatment was also negative. A small nodule had been removed from the lesion for microscopic examination and would be reported on at a future meeting.

DISCUSSION

Dr. Wise said he thought the condition was probably tuberculosis, for he could not think of anything else that it might be; but in all his experience he had yet to see a case of tuberculosis in the mouth or on the lips which was not preceded by a primary pulmonary tuberculosis. He would expect it to be shown in this case on further examination.

Dr. Whitehouse said he thought the condition was tuberculosis.

Dr. Williams said the mass was so hard and swollen that he thought it might be some process of malignant degeneration.

Dr. Schwartz said he had noted a marked induration surrounding the lesion, and he thought the possibility of a carcinoma had to be considered.

Dr. Fraser said that he was inclined to agree with the diagnosis of tuberculosis, but he thought it impossible to exclude malignancy, and suggested taking a piece of tissue for microscopic examination from the edge of the lesion

on the side of the lip where the growth was spreading. The diagnosis, in his opinion, could be settled only by histologic examination.

Dr. Howard Fox said he thought it possible that the patient had two conditions. It was well known that cancer could arise in a lupus vulgaris, and it seemed possible that this was an epithelioma complicating tuberculosis.

Arsenical Keratosis. Presented by Dr. Wise for Dr. Fordyce.

W. T., aged 35, a private patient, had psoriasis and had been taking arsenic, mostly in the form of solution of potassium arsenite (Fowler's solution), off and on for ten years. He presented a very well developed arsenical keratosis of the palms and soles.

DISCUSSION

Dr. Whitehouse said he agreed with the diagnosis, but asked how much arsenic had been given. He himself gave very little.

Dr. Fraser said he agreed with Dr. Whitehouse's remarks, and wished to know why arsenic should have been so long continued in a case of psoriasis.

Dr. Bechet remarked that arsenical keratosis seemed to occur so much more frequently after the administration of solution of potassium arsenite than after other arsenical preparations. He had observed patients who had taken other forms of arsenic for years without developing keratosis, yet he had recently noted a severe keratosis of the hands occurring in a young girl who had taken solution of potassium arsenite fairly steadily for only a few months.

Dr. Lane said that the frequency of keratosis following the use of solution of potassium arsenite was explainable by the frequency with which it was given. It was the only thing usually given by the general practitioner. No one gave sodium cacodylate injections for five or six years, and the patients did not continue these injections without advice, as they frequently did in the case of solution of potassium arsenite.

Dr. Clark said he thought it was an interesting question as to how long it takes the various arsenical preparations to produce such a keratosis. He had seen many patients with psoriasis who had been treated by an older dermatologist by intensive administration of arsenic; some of these patients had kept up the arsenic treatment for years without seeing a physician, and he had yet to see in any of these patients—some going back over twenty-five years—a single case of arsenical keratosis. One patient kept a record showing that he had taken something like 33,000 tablets of 1/30 grain (0.002 gm.) of arsenious acid, so it must be that some patients are more susceptible than others to such hyperkeratoses following arsenic.

Dr. Highman said he thought that the occurrence of arsenical keratosis in psoriasis could not be a frequent accident, or one would see more of such disturbances. It would seem to be a matter of individual susceptibility to the drug. There must be quantities of it taken throughout the country.

Dr. Lane said that for some years he had had a faint impression that the occupation of the patient who had been taking arsenic had some influence in the formation of these keratoses. The patients that he had happened to see had all been laboring people, some doing rather dirty work. He did not know whether that was significant.

Dr. Highman said he had seen the condition in persons who were not manual laborers—not many, but some. In reply to the question brought up by Dr. Fox of treating these arsenical dermatoses with the roentgen ray, personally, he would not do it.

Dr. Whitehouse asked whether there were also lesions on the soles, and said that in almost all the cases he had seen the lesions had been on the soles as well as on the palms.

Dr. Wise said that almost all the cases of this condition at the Vanderbilt Clinic revealed the fact that arsenic was taken of the patients' own volition, so that the physician was not to be blamed so much as the patient. This patient had been taking arsenic at varying intervals for ten years without consulting a physician. It might have improved the psoriasis while he was taking it. Most of the patients he had seen had taken it without prescription. As to treatment, he had but one case of arsenical keratosis in which the patient was treated with the roentgen ray—not that alone but also with salves of salicylate acid. In his opinion, the theoretical argument against roentgen ray in these cases had not much value, for the lesions of xeroderma pigmentosum often improved under roentgen-ray treatment also.

Lupus Vulgaris. Presented by Dr. Williams.

W. N., aged 25, born in Ireland, presented an eruption which began four or five months before on the left cheek as a small papule which spread quite rapidly peripherally. A crust which formed was removed by the use of an ointment. The present lesion was about the size of a half-dollar, with a slightly indurated active border showing some central atrophy. The Wassermann test and biopsy had not yet been reported.

A Case for Diagnosis: Paget's Disease? Presented by Dr. Bechet.

R. M., a man, aged 36, born in Russia, said that the lesion had been present for six months. The entire umbilicus was involved, its surface presenting a granulomatous, moist, oozing surface. A seropurulent secretion oozed from the folds around it. There was no definite ulceration, neither was there any evidence of any elevation at the borders of the lesion. It resembled somewhat a granuloma pyogenicum, yet the surface indications were suggestive of a pagetoid epithelioma. A few pustules were present immediately adjacent to the lesion, probably due to auto-inoculation.

DISCUSSION

Dr. Lane said he thought the condition was nothing but pyoderma with maceration of the surface. This condition was not uncommon in patients with the type of umbilicus seen here, which was such that cleanliness and dryness were practicably impossible. Excision was the only practicable treatment, and that should be done for the further reason of prophylaxis against the development of epithelioma.

Dr. Wise agreed with Dr. Lane, and he thought it was a form of granuloma pyogenicum.

Dr. Whitehouse agreed with Dr. Wise that it was a case of pyoderma with maceration.

Dr. Williams agreed with Dr. Lane that it was pyoderma.

Dr. Fraser said it was probably an infectious process, but one would have to think of epithelioma. A section should be taken for microscopic study.

Dr. Howard Fox said he thought the lesion should be surgically excised as there was a possibility of malignancy. A histological examination could then be made to settle the diagnosis.

Dr. Clark said he also thought the lesion should be excised immediately.

Dr. Highman said that, in addition to the other suggestions, the possibility of the first lesion being a pemphigus should be considered. It was common for it to start in the umbilicus, and he did not think it could be ruled out. He was doubtful about excising it immediately. If that were done, it would have to be practically a laparotomy, or at least as formidable an operation as in umbilical hernia.

Dr. Bechet said he agreed with all the previous opinions expressed, but was disinclined to agree with Dr. Highman's suggestion of pemphigus. The condition was too intensely localized to entertain that diagnosis.

Dr. Highman replied that, without clinging to any particular diagnosis, he had seen bullous lesions persist for months before pemphigus would be recognized.

A Case for Diagnosis. Presented by Dr. Williams.

S. F., a man, aged 40, a Russian, first presented himself in October, 1923. He had a generalized eruption which, he stated, first appeared on the lower portion of his body and gradually spread upward, the last area to be involved being the head. The Wassermann test was negative. Biopsy revealed seborrheic dermatitis. The man was referred to the dentist, who found no affected teeth. Blood chemistry revealed: sugar, 143 mg. per 100 c.c.; urea nitrogen, 22 mg. per 100 c.c.; creatinin, 1 mg. per 100 c.c.; red blood cells, 3,020,000; white blood cells, 16,000. The differential count revealed: polymorphonuclears, 72 per cent.; small lymphocytes, 19 per cent.; large mononuclears, 14 per cent.; transitionals, 4 per cent.; eosinophils, 1 per cent. The patient stated that the eruption first appeared as blisters, and that during the past week again he had noticed blisters forming. When seen in the clinic, he had never had any blisters, although the lesions appeared as though they might have been bullae which had dried.

DISCUSSION

Dr. Whitehouse said that he had not been told of the duration of the case, but it suggested impetigo contagiosum.

Dr. Bechet said that one could not be dogmatic in making a diagnosis, and that while Dr. Whitehouse's suggestion of impetigo contagiosum seemed a good one, pemphigus should also be considered. There certainly seemed to have been bullae at some stage of the eruption.

Dr. Wise said he considered the condition a type of pemphigus benignus, which was most frequently preceded by lesions simulating either seborrheic eczema or impetigo contagiosum.

Dr. Lane said that his impression was the same as that of Dr. Whitehouse. Had he seen the patient without hearing the history, he would have made a diagnosis of impetigo. It might be wise to prescribe daily scrubbing with soap and water. He doubted very much whether the antiseptic had been properly applied, and removed sufficiently often before it was reapplied.

Dr. Clark agreed with the probable diagnosis of impetigo, and said he thought the suggestions made by Dr. Lane as to treatment should be tried before considering pemphigus.

Dr. Highman said the two conditions that occurred to him were impetigo and pemphigus. There was also a possibility of bromoderma. He had seen a bromid eruption that looked not unlike this.

Dr. Williams said that he had considered practically every diagnosis mentioned. At first he thought of pyoderma and similar infections, but the condition did not improve under antiseptic treatment. The lesion under the left eye had been present since the patient was first seen. It was always active in the center, and looked like ordinary lupus erythematosus. He had thought of lupus erythematosus disseminatus with some bullous lesions, but the more he watched the case the more he thought of pemphigus, and he was inclined to believe that it would turn out to be that. The lesions were persistent; they did not appear and disappear, which would hardly be the case if it were impetigo.

A Case for Diagnosis: Lesions on the Trunk. Presented by Dr. Williams.

T. L., aged 48, an American, a machinest, was first seen in October, 1923, at which time the lesions he now presented had been present for four months. The Wassermann test at that time was one plus. Following mixed treatment for a little more than a week, a second Wassermann test taken was reported negative. The blood count showed 13,400 white blood cells. The differential count revealed: polymorphonuclears, 55 per cent.; small lymphocytes, 42 per cent.; large mononuclears, 2 per cent.; transitionals, 0; eosinophils, 1 per cent. The report of the second blood count, taken about ten days before presentation was: red blood cells, 4,000,000; white blood cells, 7,600; differential count: polymorphonuclears, 76 per cent.; small lymphocytes, 15 per cent.; large mononuclears, 1 per cent.; transitionals, 2 per cent.; eosinophils, 6 per cent. The spleen was not palpable; the left epitrochlear was enlarged, but no cervical or axillary enlargement was felt. The biopsy report was: "Inflammatory changes." The superficial changes showed a moderate amount of cellular infiltration (mononuclear), also many small blood vessels and some scattered pigment. The line of demarcation between the derma and epidermis was not well defined in some portions. There was some cellular infiltration about the sweat glands. There was basic degeneration of the superficial derma.

DISCUSSION

Dr. Fraser said that two conditions would have to be considered—parapsoriasis and urticaria pigmentosa. Pathologic examination would not aid in differentiating these conditions unless one made use of a metachromatic stain, such as thionin or toluidin blue. In the case he presented, for example, it would have been impossible to have made a diagnosis of urticaria pigmentosa from the sections stained with hematoxylin and eosin. He suggested that another biopsy be made, and that the tissue be fixed in alcohol, which brings out the granules in mast cells better than any other fixing solution.

Dr. Wise agreed with what Dr. Fraser had said.

Dr. Clark said he considered it a case of possible psoriasis. The lesions did not have much infiltration; some looked rather scaly, and there was no history of itching or irritation of any kind.

Dr. Highman said that Dr. Fraser had expressed his views. Assumming the presence of mast cells as pathognomonic for urticaria pigmentosa, nevertheless, as Hartzell pointed out, in some of the lesions in their receding stage, mast cells were absent. They were always present in the early stages, but sooner or later they were absorbed, so that in the event of negative findings for mast cells it would be well to examine two or three lesions in involution and evolution. Finally, he did not think it was urticaria pigmentosa in the sense of Nettleship, but more in the sense of refinement made by the French of the acquired or late form; possibly that variety called by them urticaria with pigment, in which mast cells were also found. Neither did the presence of mast cells characterize the lesions as urticaria pigmentosa, for these were found in other conditions in which there was local hypernutrition causing edema or lymphadenoma, such as chronic lichenification, chronic erythema—in short, wherever there had been an excess of lymph with its attendant hypernutrition. In these conditions they were not present in as great numbers as in typical cases of urticaria pigmentosa.

Dr. Fraser said that there was no other cutaneous condition in which mast cells were present in sufficient numbers to be of diagnostic value. Sometimes they were present in large numbers in nasal polyps.

Dr. Howard Fox reported on animal inoculations of ringworm of the scalp in adults. This was a further report on the two cases of ringworm previously presented. Two guinea-pigs had been inoculated, one with a culture of *Microsporon lanosum* and the other with *Microsporon audouini*. On two separate areas (in each animal) the hair was clipped and the cultures rubbed in with sandpaper. In each animal one old and one young culture was used. The culture of *Microsporon audouini*, as expected, showed no growth. In the animal inoculated with *Microsporon lanosum*, there was no result from the old (forty-three day) culture. Ten days after inoculation with the young (twenty-two day) culture an inflammatory lesion appeared, the hairs at this location showing typical masses of small spores.

Dr. Williams said he had seen the guinea-pig in the morning and saw a hair, and there was no question of the large collar with small spores.

Dr. Fraser said he also wished to be on record as having seen the slides in this case.

Dr. Clark reported on the examination of a specimen from the case of chronic lichenification shown at the December meeting. Some of the members suggested the possibility of its being mycosis fungoides, and others thought it might be a leukemic condition. The specimen was negative so far as the blood condition was concerned; neither could the diagnosis of mycosis fungoides be confirmed from the biopsy.

Dr. Wise asked whether the patient had received any roentgen-ray treatment, and said that if he had seen the patient for the first time, without the history, he would certainly give roentgen-ray treatment. The skin showed a type of infiltration which usually responded better to roentgen-ray therapy than to any other treatment.

Dr. Clark replied that there had been some rather indefinite roentgen-ray treatment over a period of two or three months, without any effect except to exaggerate the condition.

Regular Meeting, Feb. 26, 1924

W. J. HIGHMAN, M.D., *President*

ANNULAR SYPHILODERM OF THE SCALP. Presented by DR. FRASER.

A white man, aged 27, presented an eruption on the scalp of five months' duration. There was no history of syphilitic infection, but a diagnosis of annular syphiloderm of the scalp was made. The Wassermann test was twice reported 4 plus.

DISCUSSION

DR. HOWARD FOX said that he considered the lesion to be a late syphilid of the nodular type. It was not to be confused with the early annular lesions which occurred so frequently in the negro race. The somewhat unusual feature of the case was its presence on the scalp and not its annular configuration.

DR. WISE agreed with Dr. Fox that it was a late syphilid.

DR. TRIMBLE agreed with the diagnosis of syphilis, but said the lesion was what he would term a circinate nodular syphilid. He felt certain that Dr. Fraser meant that the lesion was a late manifestation of syphilis and had no connection with the annular syphilid, as we understood it.

DR. LANE said there was nothing unusual about a tertiary syphilid being annular or circinate or kidney shaped. So far as this particular lesion was concerned, it might very well be a syphilid. It also had an impetigenous appearance, but that was a common characteristic of syphilitic eruptions, especially of the face and scalp.

DR. WHITEHOUSE agreed with the diagnosis of syphilis, and also with what had been said by Drs. Trimble and Fox—that it was not an annular secondary syphilis, seen on the face and elsewhere in the colored race, and which may occur on the scalp or any where else. The lesion had improved under ammoniate of mercury, which any syphilid would do. The lesion had a nodular circinate border, without disseminated scars, and a four plus Wassermann reaction. It might therefore be considered as a late annular syphilis, not the type of the annular secondary syphilis seen in the negro. If the patient was given a full course of arsphenamin and mercury, he would probably be cured.

DRS. KINGSBURY and CLARK agreed with the diagnosis as presented.

DR. HIGHMAN said he also thought the case was one of tertiary syphilis.

DR. FRASER said he regretted that the impression seemed to prevail that he presented the case as one of early syphilis. That was not the intention. As to impetigo, Dr. Fraser failed to seen any reason for that diagnosis.

LICHEN PLANUS IN AN INFANT. Presented by DR. WISE.

J. S., a boy, aged 14 months, came to the Vanderbilt Clinic, Feb. 26, 1924. He presented an eruption of the abdomen and the anterior surfaces of the thighs, of one week's duration, consisting of numbers of distinct, shiny, waxy, papules, varying in size from that of a pinhead to one-eighth inch (0.3 c.c.) in diameter, and a few patches up to three-eighths inch (0.9 c.c.) in diameter made up of confluent minute papules. They were red, no violaceous tinge being discerned in connection with them.

DISCUSSION

DR. WHITEHOUSE said that he did not consider the condition lichen planus. Many itching eruptions of the skin would produce these papules and scratch

marks—erythema multiforme, eczema, seborrheic dermatitis and some other inflammatory diseases of the skin would produce such lesions. There was no evidence of the characteristic lichen planus papules. It seemed to be simply a pruritic papular eruption, probably due to improper feeding or to some chronic disturbance sensitizing the skin—but not lichen planus.

Dr. Fraser said he thought Dr. Wise's diagnosis should be accepted. Dr. Wise had the opportunity of studying the lesions critically and by daylight.

Dr. Bechet agreed with the diagnosis. There were distinct papules, shiny and occurring in scratch marks, like typical lichen planus. Of course, the child had also an eczema of the scrotum.

Dr. Trimble said he thought it was a case of lichen planus, although of course the patient had also an eczema on the face. He had, however, seen two negro children, of about the same age as this one, who had what seemed to be typical lichen planus, but which after a Wassermann test proved to be a miliary, flat, papular, syphilitic eruption. Dr. Fox also had presented a case before the society which looked exactly like lichen planus, and that patient also had a four plus Wassermann reaction. It would be well for Dr. Wise to rule out syphilis, although there seemed to be enough flat shiny papules to warrant the diagnosis of lichen planus.

Dr. Howard Fox agreed with Dr. Wise and Dr. Trimble that it was lichen planus. It was a difficult case in which to make a diagnosis at first glance. there were two points that spoke against eczema: the appearance of lichen-like papules occurring along scratch marks, and the circinate or "crateriform" appearance of a few lesions, a condition that was never seen in eczema.

Dr. Whitehouse protested against the diagnosis of lichen planus because of flat papules. These were seen in many dermatoses: neurodermite, erythema multiforme, scabies, and other conditions. It took more than flat topped lesions to make a lichen planus.

Dr. Clark said his first impression was that the condition was eczema, but on looking more carefully he noted what seemed to be distinct rings with shiny annular lichen papules in the circumference of the ring, and he could not connect that with a case of eczema; also he noted lichen-like papules along scratch marks, and he had come to the conclusion that although the patient was a young baby, it was probably a case of lichen planus.

Dr. Highman said that so far as he could discern, the individual, primary, unaltered lesions were of the small, waxy variety seen in lichen planus, and perhaps in lichen nitidus, lichen scrofulosorum and lichen syphiliticus. The two latter ought to be ruled out for the sake of accuracy in diagnosis; but granting that the shiny lesions were the essential ones and adding to that the presence of annular lesions, while much of what Dr. Whitehouse said was true, it would seem that by exclusion all that remained in this particular instance was the possibility of lichen planus. One should not be very much upset by the presence of what seemed to be an eczematous type of lesion, because the papules of lichen planus on that part of the body did not exclude the possibility of another lesion elsewhere. As against eczema, the absence of vesicles was a weighty factor. Lichen planus seemed to be the only plausible conception. Perhaps this was one of the rare instances in which the microscope might help make the diagnosis. Dr. Highman said he had very little respect for the microscope as a diagnostic method, but in this case it might be of value.

Dr. Wise said it might seem a poor argument to state that in an artificial light the lesion did not look so much like a lichen planus as in the day time;

but that statement held especially good in this instance, for in daylight there was not much room for any difference of opinion on the subject. Neither did he think that Dr. Whitehouse had given any good argument why the condition should not be lichen planus. The patient had shiny papules, as others had pointed out, and although he was willing to admit that one could have linear flat lesions in other eruptions, the appearance of these lesions all spoke for lichen planus. It was doubtful whether the parents would consent to a biopsy, and it might be that the diagnosis could only be made on the clinical course of the case, but he would try to report on the case later and perhaps present it again.

LICHEN PLANUS LIMITED TO THE MOUTH. Presented by DR. HOWARD FOX.

D. D., aged 23, an electrical engineer, born in the United States, had suffered from the eruption for twenty months. It consisted of superficial, grayish-white, irregular confluent patches on the mucous membrane of the cheeks and lower lip. The lesions on the buccal mucosa were mostly present along the interdental spaces. It was not possible to distinguish individual lichen papules. There were no subjective symptoms. At no time had there been any cutaneous lesions, according to the patient's statement. There was no history of syphilis, and the Wassermann test was negative. The patient had been a cigaret smoker for the past six years. Shortly after the appearance of the eruption, he had been seen by Dr. Udo J. Wile, who wrote the following in a personal communication concerning the patient. "I saw Mr. D. in 1922 with a perfectly typical lichen planus of the mouth. At that time the lesions were so typical that there could be no doubt as to the diagnosis."

DISCUSSION

DR. WISE said he thought this was another case in which the diagnosis could be made only with the greatest circumspection. It was possible to have a lichen planus in the mouth alone, but while it was exceedingly difficult to make such a diagnosis, and although he believed this to be a case of lichen planus, he was not willing to accept that as a final diagnosis.

DR. TRIMBLE said that while the lesions in this man's mouth looked very much like the other mucous lesions of lichen planus that he had seen existing at the same time with confirmatory evidence on the skin, he would hesitate to make a positive diagnosis of the lesions on the mucous membrane alone. He really did not see how one could absolutely separate the condition from leukoplakia of the same nature. He did not question the fact that if the man had a few lichen planus lesions on his arms or body, all would agree with the diagnosis; but without confirmatory evidence, the diagnosis would have to be held up until more definite evidence could be secured.

DR. LANE agreed with what had been said by Dr. Wise and Dr. Trimble, that he could see nothing that was absolutely typical of lichen planus; the fine reticulation that was so typical was not visible; it was confined to the interdental line. This was not usually the case with lichen planus.

DR. G. H. FOX said that if he had seen such lesions in connection with lichen planus on other parts of the body, he would have agreed with the diagnosis; but as it was, he was inclined to think them more like leukoplakia than lichen planus.

DR. CLARK said the lesions on the left buccal mucous membrane presented a reticulated lacelike appearance such as one does not see in leukoplakia, and he was inclined to favor the diagnosis of lichen planus.

Dr. Highman said he thought the lesions lacked the dead white chalky color which we have been led to associate with the ordinary leukoplakia; furthermore, they seemed to be discrete rather than homogeneous, and on analyzing each lesion, it seemed to be a rather dull gray spot and papule. He was not entirely reconciled to the fact that twenty cigarets a day could be responsible for the type of the lesions; in fact, if the man admitted smoking twenty cigarets, he was more likely to smoke thirty, and the lesions seemed to have a pretty clean surface. As between lichen planus and leukoplakia, he did not know on what basis the diagnosis of leukoplakia could be made, and he was inclined to the diagnosis of lichen planus. He had seen lichen planus of the buccal membrane in a woman, and from the appearance, color, shape, clinical configuration, and relation to one another, he was more inclined to side with Dr. Wile and Dr. Wise.

Dr. Howard Fox said that when he first saw this patient he was in some doubt concerning the diagnosis, and could not now be sure of it. The two conditions to be considered were leukoplakia and lichen planus. Certainly leukoplakia of this extent was uncommon in a man as young as 22. It usually occurred in persons who had had syphilis and who for a long period of time had used tobacco to excess. Neither of these conditions obtained in this patient. He made the diagnosis of lichen planus partly on its appearance and partly by exclusion. To be certain of the diagnosis it would be necessary to follow the patient until cutaneous lesions appeared, or to make a histologic examination. There was no doubt that lichen planus could exist for a varying time before the outbreak of any cutaneous lesions.

Dr. Highman said that in diagnosing lesions of this sort it was necessary to confine one's self to standards. The typical lesion of buccal lichen planus was chalky white. If there were gradations of color, he would say it was quite likely to be leukoplakia, but considering the color of the lesions in this patient, the shape, and the probability that so young a man was not likely to have leukoplakia as compared with lichen planus, the case presented seemed to be one of lichen.

Dr. Whitehouse said that there were cases independent of lichen planus, independent of smoking, and independent of syphilis; but lichen planus lesions of the tongue and buccal mucosa simulating syphilis are not uncommon. He had seen two cases in women, one a widow, with lesions of the tongue and buccal mucosa, who was treated for syphilis by a member of this society, and who would undoubtedly have been so treated by himself if he had not found circinate papules of lichen planus under the breasts and on the abdomen. After being treated for lichen planus, the lesions cleared up. She was not a smoker. In the other case, there were lesions on the mucous membrane and tongue with intense pain and the fear of cancer. The woman's husband was a syphilitic who had been extragenitally infected years before marriage. She was a highly neurotic subject, and had pains in the tongue and neck, suggesting to her both syphilis and cancer. She was incidentally seen at a time when she developed lichen planus all over the body, which she did not have before, and Dr. Whitehouse concluded that the lesions on the buccal mucosa were lichen planus. All cleared up under the treatment of lichen planus excepting the pains, for which she had been going from physician to physician. She was given arsphenamin to relieve her mind, although her Wassermann test was negative before and after the arsphenamin medication. These cases of the buccal mucosa and tongue simulating lichen planus were difficult to diagnose, and required careful consideration before reaching a definite diagnosis.

MULTIPLE BASAL CELL EPITHELIOMA. Presented by DR. TRIMBLE.

A woman, aged 57, married, and born in England, on examination presented on the skin of the back about the region of the fifth dorsal vertebra an irregularly shaped circular, scaly lesion, reddish purple, measuring one and a half inches (3.8 c.c.) in its vertical and 2 inches (5 c.c.) in its transverse diameter. The lesion was not elevated above the surrounding skin; the border was well defined, but did not have the "rolled" and "pearly" appearance usually seen in basal cell epithelioma. The lesion had appeared five years previously, but the patient could not state the length of time it took to develop to its present state.

DISCUSSION

DR. WHITEHOUSE agreed that the condition was a keratosis on the borderline of epitheliomatous degeneration.

DR. FRASER said the term precancerous dermatosis was a bad one, as it did not convey the impression of any definite clinical or pathologic picture. He agreed with the remark previously made by the chairman that histologic study in dermatology was more frequently an aid, rather than the last word in diagnosis. There were conditions, however, in which the diagnosis should rest solely on the histologic findings, and epithelioma was one of them. In this case, there was no histologic evidence of epithelioma in the sections studied. A diagnosis of epithelioma should not be made until the characteristic appearances found in that disease could be demonstrated. The clinical evidence in the case was not conclusive. The duration of the lesions was five years, yet there was no pearl-like border such as is usually seen in the so-called basal cell type of epithelioma; the lesions were flat, pigmented and scaly.

DR. LANE said he thought it was a typical case of multiple epithelioma—a very slow growth similar to the variety described by Bowen. If one or both of these lesions were taken out and enough sections studied, doubtless some epitheliomatous cells would be found in some of them.

DR. CLARK said that his impression of the lesions was different from Dr. Bechet's. The lesions did not seem to him to be a seborrheic dermatitis. He had seen a number of cases begin in this way and continue for a long time, and in his opinion these were certainly multiple, benign, superficial epitheliomas. He had noted one or two little translucent, pearly nodules, and thought the diagnosis of multiple epitheliomas was correct.

DR. HIGHMAN said that in spite of the fact that Dr. Bechet rather took the stand he himself had taken with reference to the term precancerous, it would seem that in this particular instance not only his conception, but that of most people who talk of Bowen's precancerous dermatitis, did not agree with the facts. In the first place, there were only seven authentic cases of Bowen's precancerous dermatosis on record. The cases described by Mount, Morrow, Hazen and others did not conform to this type. Whether he was fortunate in choosing the name he had for the condition he described or not, it was at least a distinct clinical entity, undergoing the changes recognized as malignant. This was not of the Bowen variety. Bowen's dermatosis looked like a tuberoserpiginous syphiloderm. One was surprised on looking at the lesions under the microscope to find that they were not unlike those of Paget's disease, with the distinct picture of basal cell epithelioma. Bowen's disease was distinctly chronic. It was not general senile hyperkeratosis.

Dr. Highman said that he agreed with Dr. Bechet that the term was a bad one. The case under consideration, however, did not belong to the Bowen group. It might be precancerous, or it might even be cancerous; but it had not the definite appearance described by Bowen. Clinically, the case presented by Dr. Trimble seemed to be a frank basal cell epithelioma, whether the particular lesions selected for study showed the necessary changes or not. On purely clinical grounds Dr. Highman thought Dr. Trimble's conception of the case was right.

Dr. Trimble said he thought it a case of basal cell epithelioma as presented. A biopsy had been made, and it would be later reported on.

A Case for Diagnosis. Lupus Erythematosus? Presented by Dr. Bechet.

R. D., a woman, aged 33, born in the United States, said that the lesion on the nose had been present for seven years. The infiltrated plaque on the right cheek and the pernio-like lesions on the hands had appeared three months previously. On the nose, was a typical scaly, sharply marginated patch of lupus erythematosus. On the right cheek, there was a rounded, raised, quite red, markedly infiltrated lesion of a boardlike hardness. This lesion was about an inch (2.5 c.c.) in diameter, and the infiltration was subcutaneous. The dorsal surfaces of the hands and phalanges were covered with an aggregation of large, violaceous red, conglomerate patches with slightly scaly surfaces. These lesions burned and itched considerably.

DISCUSSION

Dr. Wise said he thought the case showed all the characteristics of lupus erythematosus.

Several of the members agreed with him.

Dr. Fraser said he thought the condition looked like an early case of lupus erythematosus.

Dr. Whitehouse said the case brought up a subject in which Dr. Clark was very much interested—the relationship if any, of lupus erythematosus to tuberculosis. The woman had nodular lesions, and the lesions on the hands were of that character. In a similar case of his, there was a group of nodular lesions on the side of the neck with typical lupus erythematosus of both cheeks. The young man said that the lesions on the cheeks had been treated by roentgen ray, and large scars were present on the left cheek. The remains of lupus erythematosus on the right cheek, with the nodular lesions on the neck, were still present; it was almost similar to the case presented. Such cases were suggestive, and while the case under consideration appeared to be lupus erythematosus, it was impossible to say whether or not it was associated with tuberculosis. The etiologic factor in the case preceding the whole condition was problematic. So many cases of lupus erythematosus end in pulmonary, osseous, or kidney tuberculosis, that it was evident that our knowledge of the subject was still confused.

Dr. Bechet said the patient presented a sarcoid-like lesion on the cheek a typical lupus erythematosus on the nose, and lupus pernio on the hands. There was no doubt that these lesions were simply different manifestations of the same disease, namely lupus erythematosus. It was the occurrence of just such cases which awoke the suspicion that lupus erythematosus belonged to the group of dermatoses allied in some way to tuberculosis.

A Case for Diagnosis: Circinate Lesion on the Face. Lupus Erythematosus or Syphilis. Presented by Dr. Bechet.

D. B., a woman, aged 44, said that the lesion had been present for one year. Two Wassermann tests had been made, six months, and three weeks previously; both were negative. No specific history could be elicited. The lesion was slightly scaly and markedly circinate in character. There was some suggestive atrophy in its center, and no tendency to ulcerate.

Dr. Bechet said that in view of the history of two negative Wassermann tests, and the appearance of the lesion, he was more inclined to make the diagnosis of lupus erythematosus. He would give her some specific treatment, and report on the results attained at the next meeting.

Lichen Planus, Localized, in a Negress. Presented by Dr. Howard Fox.

T. J., aged 24, married, a full-blooded negress, had suffered from the eruption for six weeks. It was situated on the lower two thirds of the postero-external aspect of the left thigh. It consisted of numerous groups of small flat papules of a darker color than the normal skin and giving a suggestion of a violaceous color. There were moderate infiltration and slight scaling. No scratch marks were noted, although the patient complained of moderate itching. There were no lesions in the mouth. The patient was a well nourished woman in apparent perfect health.

Note.—A histologic examination made by Dr. J. Frank Fraser subsequent to presentation showed the typical structure of lichen planus.

DISCUSSION

Dr. Wise said it was another case in which the diagnosis was difficult, even more difficult than that of the baby he had shown with lichen planus. This patient also had evidence of lichen planus; on the other hand, the condition looked more like neurodermite. He did not feel that he could offer a diagnosis, as it rested on the microscopic diagnosis to decide.

Dr. Trimble was not willing to accept an absolute diagnosis of lichen planus. Like Dr. Wise, he had not noted any distinct individual lesion typical of lichen planus, and the fact that it was on only one side would seem to speak against that diagnosis, although unilateral lichen planus may exist. As there were no typical lesions and as the condition was confined to one thigh, it was possible that the disease might be tuberculosis. He then recalled the case of a young woman presented some years ago by Dr. Howard Fox with a similar lesion on the thigh. Dr. Fox later published an account of some unusual types of tuberculosis which he read before the American Dermatological Association, in which was incorporated the case mentioned. The configuration of the eruption was just like that in the present case, although it had distinct papules, which were not seen in this case. It might be an unusual type of tuberculosis, similar to the one shown before, scaly and papular and confined to one thigh. A positive diagnosis, however, would have to be held up for further examination.

Dr. Fraser expressed regret for not having a report on the histopathology. It would be presented at the next meeting.

Dr. Clark said that lesions of the kind in a colored person were so confusing that he could not venture a diagnosis.

Dr. Highman said that it seemed to him to be a case of hyperpigmentation with a slight tendency to papular elevation, and that he did not know of any type of lesion excepting a syphiloderm or lichen planus which would cause quite that picture. Lichen planus leads to hyperpigmentation as the lesions resolve, and this, seemed to be the most likely diagnosis. The fact that the lesions were polygonal seemed to be further evidence of lichen planus, and it appeared probable that Dr. Fraser's histologic examination would furnish final corroboration.

Dr. Howard Fox said that while favoring a diagnosis of lichen planus, he admitted the case to be an atypical one. Dr. Trimble's suggestion of a similarity to one of his cases of superficial tuberculosis was of interest. The speaker recalled his case, and admitted the similarity both of appearance and location.

Disseminated Lupus Vulgaris. Presented by Dr. Wise.

N. J., a man, aged 22, colored, single, a porter, came to the Vanderbilt Clinic, Feb. 14, 1924, complaining of an eruption of four months' duration. There were no subjective symptoms present. He presented an eruption involving the lower eyelids, the forehead, nares and the lips near the angles of the mouth. On the eyelids there were discrete and confluent raised papules varying in size from that of a millet seed to that of a small pea. On each eyebrow there was a millet seed sized wart. The angles of the mouth presented several small verrucous lesions together, with split papules in the right corner. The nares were greatly thickened and showed lesions consisting of shiny papules varying in size from that of a millet seed to that of a small pea. On the forehead, near the hairline, there was a small circinate erythematous crusted lesion. The patient denied syphilitic infection, and the Wassermann reaction was said to have been negative four months before. The Wassermann report at the Vanderbilt Clinic showed a one plus alcoholic antigen and a four plus cholesterin antigen reaction. The case was analogous to four or five cases which had been described by Dr. Fox, all of which, however, occurred in females. Dr. Wise said that this was the first case of the kind that he had seen in a male, and he presented the case with a tentative diagnosis.

DISCUSSION

Dr. Whitehouse said that Dr. Wise had stated the case better than he could, but he did not see why it should not be considered a case of circinate nodular syphilid, before deciding on the diagnosis of lupus vulgaris. If that had not already been eliminated, it should be considered.

Dr. Trimble said that the fact that this patient had what one might call a granular condition of the mucocutaneous junction of the nostrils made it look very much like a cutaneous tuberculosis. The entire lesion, however, taken in connection with the four plus Wassermann test, inclined him quite strongly to the diagnosis of syphilis. One sees sometimes what might be called a tertiary syphilitic nose, very much pock-marked and with punctate scarring due to syphilis, and this might be a beginning case of that type. From the clinical standpoint, he would call it syphilis, reserving the right to change his opinion after the report of the microscopic examination and therapeutic test.

Dr. Howard Fox said he thought this was another case in which the clinical diagnosis was difficult. He favored Dr. Wise's diagnosis of lupus. The two

conditions to be considered were lupus and syphilis. If this was syphilis, it was of the late type in which the lesions tended to be more or less asymmetrical. In this case, the nodules were scattered over the face without particular grouping, and were equally profuse in number on both sides of the nose. One of his patients with miliary lupus had suffered also from syphilis, antisyphilitic treatment having no effect on the eruption.

Dr. Clark said that while it was generally believed that tertiary lesions were asymmetrical, exceptions to this were frequent in syphilitic lesions of the face. In many cases, both alae were involved. He would also suspect lesions that occurred at the angles of the mouth; that, together with the four plus Wassermann test, inclined him to the diagnosis of syphilis, and he would advise intensive treatment for a while at least before pronouncing the condition malignant.

Dr. Highman said that the condition was either syphilis or tuberculosis, and that a therapeutic test was indicated, if not a biopsy.

Dr. Wise said that a report of the microscopic examination would be submitted at the next meeting.

Dr. Whitehouse, referring to the clinical features of syphilis, said it was conceded by all that they simulated almost every known dermatologic lesion of the skin, including lupus. Referring to Dr. Clark's remarks about tertiary syphilis being usually confined to one side of the body, he did not think that these exceptions applied to the face. It had been taught that tertiary syphilis of the arms, hands, etc., was usually on one side, but he had seen a number of instances in which it was on both sides, so it could not be said to be unusual. It was highly important that the case should be presented again after the patient had received antisyphilitic treatment.

Dr. Clark said that he hoped to present a patient of whom he had spoken before, a young man with xanthoma diabeticorum, who had been discharged from the hospital after the disappearance of all his lesions. He was rather an irresponsible person and had given up the use of iletin or insulin, and it might not be possible to get hold of him again. The lesions when examined were full of cholesterol, and the blood was unusual in character; from the amount of fat in it it had almost a milky appearance. He had over 500 mg. of sugar in his blood, and almost a solid urine. The urine cleared up promptly in three days under iletin medication, and his symptoms and headaches gradually improved; but it was three weeks before much change occurred in his skin lesions; eight weeks of treatment had cleared up most of the verrucous-like tubercles. Photographs were exhibited of the original condition.

Dr. Bechet reported that the patient presented by him at the last meeting of the Society as having a case for diagnosis; a nodular, ulcerating lesion of the upper lip with total loss of tissue resembling a hair lip, had had a small section of the lesion excised, and the pathologic report had been prickle cell epithelioma. At the time of presentation it had been the consensus of opinion that the lesion was tuberculous. He himself had agreed with that diagnosis, and in his opinion the histologic diagnosis did not disprove the clinical one, as it was well known that epithelioma occasionally began on tuberculous tissue, as notably evidenced in lupus vulgaris. This might hold true in the case under consideration. The material for the biopsy consisted of a small nodule from the upper margin of the lesion.

NEW YORK ACADEMY OF MEDICINE, SECTION ON DERMATOLOGY AND SYPHILIS

Regular Meeting, Feb. 5, 1924

PAUL E. BECHET, M.D., *Chairman*

FIXED ERUPTION FOLLOWING ARSPHENAMIN MEDICATION. Presented by DR. CHARGIN.

G. R., a man, aged 40, had a syphilitic history which dated back about twenty years. During the past few years he had been under treatment at the Mount Sinai Clinic, receiving thirty-one arsphenamin injections in addition to fifty-eight injections of mercury. Following the thirtieth injection of arsphenamin, the patient developed circular erythematous patches on the neck, which as they cleared up left the typical pigmentation seen in the fixed eruptions. The eruption appeared within twenty-four hours following the injection of arsphenamin. The pigmentation had almost faded out when three days before presentation the patient received a further injection of arsphenamin. Twenty-four hours later the old lesions reappeared, showing an increase in the erythema at the periphery of the old lesions, and a few new lesions appeared under the chin. The patient had taken phenolphthalein on many occasions during the past year, but had never noted that an eruption had followed.

DISCUSSION

DR. GILMOUR said the condition looked like an Ex-Lax eruption, but as there was nothing in the history to support this diagnosis, the diagnosis would have to be accepted as given.

SARCOID. Presented by DR. WALZER for DR. LAPOWSKI.

S. S., a woman, aged 23, single, was deaf and dumb. Her father died of tuberculosis of the lung six years ago. This was the only thing of importance in the family history. When 11 months old, the patient had had an attack of meningitis, followed by permanent loss of speech and hearing. When 12 years of age, a tuberculous gland of the right side of the neck had been removed. The scar was still present. The patient claimed she had been well since. The present trouble started with a pea-sized, red, elevated spot on the cheek about fifteen months before presentation; this increased to the size of a quarter, and finally disappeared about two and a half months ago. About seven months after the appearance of the first spot, another pea-sized spot appeared on the bridge of the nose and had increased since. Two weeks ago another spot appeared on the left index finger. The woman was first seen at the Good Samaritan Dispensary on November 15. She then showed on the left cheek a quarter-sized annular patch, slightly depressed, of a faint pink color, in the center of which was a brown pea-sized spot. On the nose was a one cent-sized erythematous, elevated patch with a deep center. There was no scarring or atrophy. On diascopic pressure, tiny pigmented spots were seen. At present, she showed on the base of the left index finger, dorsum, a one cent-sized, erythematous patch, not sharply outlined, elevated, which disappeared completely on pressure. This was of two weeks' duration. On the upper part of the nose was a one-half dollar-sized, erythematous, annular

ring, sharply outlined, the edges elevated, and the ring not completely closed at the upper left part. The center of the ring was pale, depressed, looked atrophic, and gradually merged into a pinkish border. On diascopic pressure, the border in some places showed dark brown points. To the left of this lesion was a pea-sized depressed scar, the remains of an injury six months before. On the left cheek was a dollar-sized, faint, whitish patch, which looked like a faint atrophy, with the edges more pronounced, very faint, pink and slightly elevated. Physical examination revealed old fibrous lesions of the right upper lobe and suspicious signs of the left apex (tuberculous?). The roentgen-ray report showed negative findings of the lungs.

DISCUSSION

Drs. Pollitzer and Rothwell agreed with the diagnosis.

Dr. Wolf did not understand how from the appearance of the lesion any one could make the diagnosis of sarcoid, and asked how the lesion looked when it was first seen.

Dr. Fraser agreed with Dr. Wolf, and said the lesion looked more like lupus erythematosus than sarcoid.

Dr. Walzer said the patient had been under observation for some time, and some of the lesions were seen developing. The lesion on the nose first appeared as an infiltrated dime-sized erythematous patch. There was no scaling, no patent follicular openings. It gradually increased in size with ring formation, the center becoming depressed, covered with a thin, easily removable scale, but no scales were present on the still active part of the lesion. This patch was now gradually disappearing. The lesion on the finger appeared in the same way, just as an erythematous patch, infiltrated, no scaling, no patent follicular openings. With the disappearance of the lesion, there was a faint, scarcely perceptible scar (on the left cheek).

Scleroderma. Presented by Dr. Rothwell.

G. R., a woman, aged 27, single, born in the United States, presented on the face, chest and hands a hidebound condition of the skin. On the face was to be observed the tight, immobile expression of the mouth, and the hands (fingers) showed sclerodactylia. The unusual part of the picture was a sprinkling of the affected parts with numerous small (pea-sized and smaller) spots of erythematous character, disappearing on pressure, to return again shortly, which the patient said had appeared after injections administered some time previously in the course of her treatment elsewhere. The condition had been of six years' duration.

DISCUSSION

Dr. Pollitzer said the case presented by Dr. Rothwell was an extensive case of scleroderma with the unusual feature of a disseminated network of dilated blood vessels. Of course, in scleroderma there was intereference with the circulation, and it was conceivable that the interference might be of such a nature as to block up the vessels at one point and at another point to cause a dilatation, but this was certainly an unusual appearance in scleroderma.

Dr. Rothwell said he was glad to have Dr. Pollitzer's suggestion about a reason for the eruption and erythema—the telangiectasia, he called it.

SCLERODERMA. Presented by DR. WALZER for DR. LAPOWSKI.

E. R., a schoolgirl, aged 10, was the second of a family of five, the remaining four being normal. The trouble began about a year and a half before on the thigh as itchy "white spots." One year before, a patch appeared on the right leg, and the skin on the abdomen became shiny. Six months before, lesions began on the right elbow. There was a history of one abortion in the mother before the birth of the patient, and two after. The child never was sick. At present she showed: On the abdomen, a faintly outlined irregular patch from the ensiform cartilage down; the follicular openings were prominent and the skin slightly infiltrated. The thighs, front and back, and both legs, showed irregularly shaped patches; certain parts within were ivory colored, hard, infiltrated and smooth. The remaining portions of the skin were somewhat violaceous and atrophic. The veins showed through prominently, and the skin was softer than in other parts. On the right side of the body, in the axillary line, was one irregular, oval, pigmented patch, brownish, smooth and not infiltrated or atrophic. On the outer surface of the right elbow were numerous pea-sized, whitish, sharply outlined, slightly infiltrated patches, with no inflammatory zone. In the center was a faint, irregular, slightly elevated, infiltrated, reddish patch. This was probably an erythematous patch undergoing sclerotic changes. The nails, face and scalp were normal. The Wassermann test of the mother of the patient was negative.

DISCUSSION

DR. WALZER said that there was a distinct scleroderma on the thighs, but the most interesting lesions were those on the right elbow. There was a group of about a dozen or more pea-sized whitish atrophic spots. In the center of this group was a pea-sized, erythematous slightly infiltrated lesion with no areola, which in all probability was the forerunner of these atrophic spots. These atrophic spots alone, without the skin condition, could be called "white spot disease," but occurring in a patient with scleroderma, they were no doubt atypical spots of circumscribed scleroderma; in other words, "white spot disease" can probably be put either in the lichen atrophicus family or in the scleroderma family.

MILIARY LUPUS. Presented by DR. WALZER for DR. LAPOWSKI.

E. O., a girl, aged 5, showed nothing of any interest in the family history. She had never had an infectious disease. About one year before presentation, the child developed enlarged glands of the neck, and seven months before the tonsils were removed. According to the mother's story, the patient had been in fairly good health up to the present illness. The present condition developed nine weeks ago, with "red spots" on the back, chin, and upper extremities. Six weeks ago, the mother noticed similar lesions on the buttocks and lower extremities. About two weeks ago, the child injured her mouth with a bottle, and two or three days later the lips became sore and swollen. During the last two months the child had lost 10 pounds (4 kg.). The patient did not have a cough or night sweats. She had lesions on the arms, right cheek, left corner of the mouth, back of the left ear lobe, right leg and thigh and left leg. The primary lesion was a tiny pinpoint to pin-head sized papule, red and slightly elevated, either scattered singly, or forming patches. These patches varied from the size of a pea to that of one cent, were red, sharply

outlined, and covered with thin, adherent crusts. On removal of the crusts, individual follicular papules could be seen. On healing, these lesions left pitted scars. On the left foot, externally, there was a plaque composed of two patches, one cent sized, erythematous, sharply outlined; the entire plaque was covered with a thin, whitish, adherent crust. In the center of the upper lip were a number of pinhead-sized pustules on an erythematous base. In the center of the lower lip was one pea-sized ulceration. The condition had greatly improved since first seen. (The mother claimed the condition followed trauma.) The submaxillary, axillary and inguinal glands were palpable. Physical examination revealed suspicious signs in the left apex. The roentgen ray showed no evidence of lung lesions. The Pirquet test was negative.

DISCUSSION

DR. POLLITZER said he hesitated to discuss the case because he differed absolutely as to the diagnosis; but he felt at a great disadvantage in having seen the case for only three or four minutes in a poor light as against the ample opportunity with daylight which the presenter had had. Subject to correction, however, he would say there did not seem to be anything of a tuberculous process. The lesions were not infiltrated, but were superficial papules—some of them barely that, some merely macules—that seemed to be the faded remains of former lesions. The large one on the left leg had a soft crust like that from exudation following a bromid eruption; and on inquiry the mother said that they appeared at one time as raised red lesions which discharged pus; and the one on the leg which looked like a bromid lesion, she described perfectly as a large bromid nodule.

DR. WALZER said this case was presented because of the tiny pinhead sized follicular papules. These softened, with no pus formation, and soon became covered with a thin, adherent, dry scab. On removal of the scab the surface was dry, but soon bleeding points appeared. There was no inflammatory areola. Tiny superficial scars were left. Many of these follicular lesions had united into one patch, and on removal of the scab there were a number of tiny follicular depressions which eventually left a patch of tiny scars. No drug was taken internally which would cause a picture like this; besides the mother of the child was positive that no drugs had been taken for about one year. The lesions were not those of a simple infection as was suggested, nor could tiny grouped follicular lesions be produced by scratching.

TUBERCULID. Presented by DR. WALZER for DR. LAPOWSKI.

E. W., a girl, aged 4 years and 6 months, whose family history was not important, had been vaccinated when 8 months of age. The lesions first appeared when the child was 14 months old. With the exception of the abdomen and chest anteriorly, almost the entire body was affected. At present she showed: (1) numerous scattered flat, papular, follicular lesions that became necrotic and left scars; (2) scattered, ulcerations not very superficial, impetiginous, sharply outlined, surrounded by an erythematous halo; many covered with crusts not firmly attached, which on removal showed the base to be composed of numerous follicular lesions; (3) scattered scars over almost the entire body. These were the result of the lesions. The scars were all sizes and shapes, some pinkish, most of them white, sharply outlined, depressed, some with serpiginous borders. (4) The lower lip showed a whitish, scarring patch, hard, infiltrated, with two pea-sized, reddish ulcerations. The only

positive findings from the physical examination were enlarged cervical glands, large diseased tonsils, and a slightly enlarged liver. The Pirquet test was positive, the Wassermann test negative. The roentgen ray showed normal chest, lungs and glands.

DISCUSSION

DRS. SCHEER, CHARGIN and GILMOUR agreed with the diagnosis.

DR. ABRAMOWITZ said that the lesion did not seem to be either tuberculosis or tuberculoid in nature. There was a definite history of the eruption appearing about six months after the child was vaccinated. The eruption was recurrent and of a vesicular nature, leaving crusts, and resembled postvaccinal pemphigoid dermatitis.

SYCOSIS PLUS ROENTGEN-RAY THERAPY. Presented by DR. WALZER for DR. LAPOWSKI.

A. S., a man, aged 24, single, with a family history of no importance, had a condition that began about seven years before presentation, in the bearded region. For two years he was treated in the Good Samaritan Dispensary and various clinics with local applications of all sorts, and vaccines. His face finally cleared up after about twenty-seven roentgen-ray applications given in one of the city clinics. A short time later, the patient returned to the Good Samaritan Dispensary, with a complete recurrence. No local treatment seemed to make any impression on the lesions. The man finally went to the Metropolitan Hospital, and was there for six months. Manual epilation of all the hair and local application of salves improved the condition; but the disease soon broke out on the thighs and lower extremities and relapsed again on his face. He returned to one of the city clinics and received more roentgen ray treatment on his face and extremities, and after numerous relapses of severe inflammatory, oozing, impetiginous lesions, he returned to the Good Samaritan Dispensary about one year after the last visit, with the skin on the bearded region smooth, atrophied, reddish, and with practically no signs of any active process. The thighs, however, still showed active lesions. This was in January, 1923. In about April, 1923, he again returned to the Good Samaritan Dispensary with a complete recurrence of a severe aggravated follicular sycosis of the face, thighs and legs. He returned to the Metropolitan Hospital where he received about fifteen injections of autoserum, nine arsphenamin injections and local treatment. He remained there for eight months, and was discharged about the middle of last December (almost two months ago) without much improvement. At present he showed about the bearded region almost complete atrophy and some telangiectasia; about the chin, upper lip, eyebrows, eyelashes and neck, there were red, infiltrated, crusted patches of sycosis; from the symphysis pubis down to the toes the skin was completely covered with papular follicular lesions. The skin was atrophied. The Wassermann test was negative.

DISCUSSION

DR. WALZER said that this case was shown not so much for diagnosis but to show the result of treatment. The man received at first about twenty-seven roentgen-ray treatments in succession at about weekly intervals, and the spreading of the disease occurred after these treatments. These deep-seated, follicular sycoses would eventually improve with local treatment. The roentgen-ray, given in such quantity as was received by this patient, was undoubtedly a great factor in the spread and aggravation of the disease.

Dr. Pollitzer inquired on what basis the diagnosis of tinea sycosis was made. In his opinion, the case was one of common coccogenetic folliculitis.

Dr. Wolf agreed with Dr. Walzer that the prolonged use of the roentgen ray had a deleterious effect on the entire organism. If a patient had a localized infection, the long continued use of the roentgen ray diminished the resistance of the entire epithelial surface, and unexposed parts of the skin that were perfectly healthy, as the result of this diminished resistance had a tendency to infection. The case brought out this point strikingly. Dr. Wolf said that the use of the roentgen ray without limit, as practiced in various parts of the city, was a great injustice. Many men did not know when to stop using the roentgen ray for various dermatologic conditions, but continued its use until radiodermatitis, and other conditions, appeared. One should bear in mind the fact that other conditions did result as a by-effect of continued roentgen-ray treatment.

Dr. Walzer said the diagnosis originally made about seven years before was purely on clinical grounds.

A Case for Diagnosis: Sarcoid? Fibroma? Presented by Dr. Bechet.

H. D., a man, aged 27, born in Russia, said that his lesions appeared one year previously. There were probably eighteen or twenty small, rounded, subcutaneous tumors, about one-quarter inch (0.6 cm.) in diameter, and irregularly distributed over the trunk, arms and legs. They seemed to be attached to the overlying skin, and were very hard to the touch. The patient said that they were painless. Two of the tumors had been removed for microscopic study, and a report of the result would be made at the next meeting.

DISCUSSION

Dr. Bechet said that the diagnosis of sarcoid seemed the most plausible, but it was impossible to make a definite diagnosis without microscopic study. He hoped to make a more definite report at the next meeting.

Dermatitis Exfoliativa (Lymphatic Leukemia). Presented by Dr. Walzer for Dr. Lapowski.

P. DeM., a man, aged 62, married, said that about thirty-five years before he had had a tooth extracted and his jaw was broken; nothing else in his past history was of any importance. The present trouble began a year before presentation, with itchy palms, and the itching gradually spread over the entire body. At present the entire body was a peculiar dusky red, the skin was slightly thickened, dry, pruriginous, with a mild accentuation of normal lines and covered with scales; in front the scales were furfuraceous, and in back they were thick, attached in the center, with the edges free. On the hands and feet, the skin was thick and covered with rhagades. The nails were thick and heaped up at the edges. The scalp was covered with thick, whitish scales. All the glands were markedly enlarged, not very hard, some clumped, some single. Physical examination was negative. There was no enlargement of the liver and spleen. A number of blood examinations revealed: white blood cells, about 10,000, with a lymphocytosis varying from 62 to 78 per cent.; red blood cells, normal. The urine showed urobilinogen + + +. The Wassermann test was negative. From Oct. 9 until Dec. 11, 1923, the patient received five injections of neo-arsphenamin intravenously and sulphur ointment locally, with

practically no improvement in the condition. Roentgen-ray examination of the chest showed the right hilus infiltrated; it might be secondary to leukemia.

DISCUSSION

DR. MCCAFFERTY referred to an article recently published by Dr. Ketron. In this article, he said that he could find but five other cases of lymphatic leukemia reported in the literature. The white blood counts usually ran from 30,000 to 200,000 or more, there being a great increase of either large or small lymphocytes. In most of the five cases there had been an increased number of large lymphocytes; whereas in Dr. Ketron's case, the small lymphocytes were increased. In the patient whom Dr. Walzer presented, the hair had fallen out, the finger nails were greatly thickened, and there was an exfoliation of the skin which compared somewhat to Dr. Ketron's case; but there was no infiltration in Dr. Walzer's case, like that seen in Dr. Ketron's case. The white blood cells in the case presented by Dr. Walzer numbered only 10,000, which would exclude the possibility of lymphatic leukemia. It is possible that the case might be one of a lymphatic leukemia, which had been described fully by Dr. Wise.

DR. POLLITZER asked how many blood counts were made and was told that there had been four: 10,000, 12,000, etc. Dr. Pollitzer replied that that was a very low count for lymphatic leukemia.

DR. LEVIN said that so far as the blood count was concerned, it might be a case of subleukemic leukemia. Dr. Ketron's case was a true leukemia of the skin with the characteristic leukemic infiltrations. The case presented might be a leukemid.

DR. WALZER said leukemia was a disease of the blood forming organs and that the blood picture was only a symptom of a disease. This case presented a blood count of 68 to 72 per cent. lymphocytes and a leukocytosis of 10,000 or more. In other words, it was a relative lymphocytosis, not an absolute. Blood-forming organs at this particular time did not throw off an excessive number of cells, so that there was not an absolute lymphocytosis in the blood; however, during some time later in the course of the development of this disease, the patient will probably show an absolute lymphocytosis, but this was not really necessary for the diagnosis of the disease. The case was an erythroderma exfoliativa, universal, aleukemic leukemia.

FIVE CASES IN WHICH RADIUM TREATMENT WAS USED. Presented by DR. TRIMBLE.

Tuberculosis Ulcerosa.— A woman, aged 20, born in Italy, had an ulcerated, warty area on the margin of the left ala of the nose, which had existed for three months. There was a slight superficial ulceration on the mucous membrane of the same side. Radium was applied for five and one-half hours with one-half strength plaques.

Epithelioma of Nose.—A man, aged 38, born in the United States, had a lesion that covered practically the whole nose, and there was one other lesion on the left temple. The latter lesion was about the size of a five cent piece. The duration was fourteen years. The depression was due to former treatment with carbon dioxid snow. The treatment consisted of eleven hours of radium application, which resulted in bringing the lesion to its present condition. The crust covering one unhealed area had received treatment with

radium for hours without result. The lesion on the temple showed a good result, although the disease still existed in the upper border.

Keloid.—A man, aged 23, born in the United States, had a keloid that was idiopathic or spontaneous in nature, situated on the back of the neck. The size was that of a silver quarter and protruded about one-half inch (1.2 cm.). The treatment consisted of the application of radium for twelve hours with one-half strength plaque. The result was considered good, although the pigment was removed.

Angiomatous Nevus.—A girl, aged 1, had a lesion that was situated on the upper part of the chest, just a little to the left of the median line. It was brilliant red, about the size of a pigeon's egg, and was raised above the surface from one-half to 1 inch (1.2 cm. to 2.5 cm.). The treatment had consisted of the application of radium with one-half strenght plaque for seven and one-half hours.

Epithelioma of the Lip.—A man, aged 40, born in the United States, had a lesion situated on the left side of the vermilion border of the lower lip. It had the characteristic rolled border and all the clinical evidences of the disease in question. Radium was applied, with a one-half strength plaque, for eleven hours.

DISCUSSION

Dr. Trimble said the cases were presented because the Section might be interested in seeing a variety of lesions showing the effect of radium treatment. In his opinion, some of the results were striking, especially in the child with angioma on the chest. He was particularly interested in provoking some discussion on the case of epithelioma of the vermilion border of the lip. While he did not consider the patient cured, those who had seen the photograph taken before treatment could see the marked improvement. When last examined there were no enlarged glands in the neck, and it seemed as if recovery were assured. The young man with the epithelioma on the nose showed a good result of treatment, except at one point where the lesion had not completely healed. Dr. Trimble said he was considering whether to proceed further with the radium or to change the method of treatment.

Dr. Wise, referring to the case of epithelioma of the lip, said he thought it was justifiable to treat the patient with radium, providing sufficient attention was paid to possible metastases. Angioma was treated satisfactorily with radium. With the moderate amount of radium which he possessed, he had obtained excellent results in these cases. The patients that Dr. Trimble presented showed good results, with the exception of the one with the case of epithelioma of the nose, who would probably get well under curettage followed by cauterization.

Dr. Rulison said he thought the results obtained by Dr. Trimble were satisfactory, but he wished to know more of the details as to how the patients were treated—the distance between plaque and skin, whether filtered or not, and the intervals between exposures. It is important in these patients treated with radium that we should know exactly what was done, what the time interval was, etc., for these details make all the difference in the world in the results.

Dr. Wertheimer, referring to the case of epithelioma of the lip, said that when these cases were treated, it was best after treating the lesion with radium to be sure to irradiate the glands in the infected region. The point brought

out by Dr. Wise about following up the radium and roentgen-ray treatment for fear of metastases was important. Another point in the use of radium was in regard to plantar warts. He had always had excellent results by exposing the wart for one and one-half hours to a 10 mg. plaque, unscreened.

Dr. Trimble, referring to what had been said about following up the treatment, said that the consensus of opinion was that roentgen rays should be used on the glands of the neck, but that he was not so sure of this. So far as could be clinically determined, the glands in the neck were not affected, and it was sometimes best to let sleeping dogs lie. Excision, however, could be easily performed if it were thought necessary. This was the only patient with epithelioma of the lip that he had treated with radium, and the man was still under treatment. No screening was done in any of the cases shown, only a few layers of rubber dam being placed over the applicator. The milligram hours were merely counted up, some exposures were as long as two hours and some as short as forty-five minutes. The exposure time was added up into milligram hours. In the case of the epithelioma on the nose, it did not seem possible to curet the growth when the patient first presented himself. The entire nose was affected, and it did not seem wise to curet the epidermis from the entire nose. In other words, the case did not seem amenable to that form of treatment, so it was decided to begin with radium. The unhealed portion of the lesion could be curetted now, but Dr. Trimble said that he hesitated to do this because he had in former times curetted parts that had been irradiated with the roentgen ray, and the result was not good, as the tissue crumbled in a most peculiar way, the healing was slow, and the sloughing was considerable. He thought the same condition would probably exist with radium. His results with basal cell epithelioma had been good, but with the squamous cell epithelioma he had been unfortunate in a number of cases. One basal cell epithelioma, after apparently having been healed for a year or more, had recurred, showing epitheliomas did recur after radium treatment.

Dr. McCafferty, referring to plantar warts, said that he had had good results from the use of the roentgen rays. He preferred using unfiltered radiation, employing ½ units to each wart, shielding closely at the first treatment. Treatments should be repeated in from four to six weeks. It usually required two or three treatments to eradicate plantar warts completely. He believed that the roentgen rays had the advantage over radium because they were quicker, and the dosage could be measured more accurately. Callosities responded to the roentgen rays with the same form of treatment, but were occasionally a little more resistant.

Syphilis of Yaws. Presented by Dr. Thornley.

R. R., said that his mother had died of cancer of the breast. He had had gonorrhea several times. His present illness began six months before presentation, with a sore on the penis which appeared eight days after the last intercourse. This sore healed in four weeks under local treatment only. No Wassermann test or dark-field examination was made at this time, and no eruption was noticed. The patient suffered from slight headaches, worse during the day, and occasional sore throat. Eighteen days ago, the eruption appeared on the face and scalp. He had no headaches and no digestive disturbances or urinary symptoms. The dark-field examination of the eruption on face was positive. The Wassermann test was positive. The pupils reacted to light.

The left epitrochlear gland was larger than the right. The mucous membranes were negative. There were several vegetating papules on the face and scalp. There was a macular roseola of the trunk.

DISCUSSION

DR. POLLITZER said he thought it was a case of syphilis. The man had a penile lesion, or had had one six months before. Now he had moist papules on the face which of course were loaded with spirochetes. Dr. Pollitzer said that he did not know how the examination was made, but that there were differences between the organisms of yaws and syphilis which it was possible to recognize. The diagnosis, of course, was between syphilis and yaws. The fact that the man had been in the tropics perhaps suggested yaws. It would be interesting to hear the argument in favor of the diagnosis of yaws. To him the case seemed to be evidently a case of syphilis.

DR. GOODMAN said he thought the case was one of syphilis. Dr. Pollitzer had cited sufficient reasons, and in addition the man presented a generalized eruption of ordinary macular syphilis. The fact that the man had been in the tropics did not indicate that he had yaws, for men in the tropics also had syphilis and presented multiform eruptions as this man did. It was difficult to see much that was closely allied to yaws, either as Dr. Goodman had seen the disease in the tropics, or as it had been presented before the Section. The lesions were nearly the color of the man's skin, and not as yellowish as the crusts of frambesia tropica. He would have liked to see the lesions under the crusts, for in yaws it is possible to see the raspberry-like lesions from the appearance of which the disease received its name. The finding of spirochetes in the lesions was not conclusive one way or the other. It was possible to find *Spirochaeta pallida* in the moist papular type of syphilis, and that might be the case here. As for differentiating *Spirochaeta pallida* from *Spirochaeta pertenuis* of yaws under the dark-field microscope, the task was not easy, if at all possible. Dr. Goodman had discussed at the last meeting of the Section the procedures which led to the differentiation of these spiral organisms by laboratory methods of the clinical laboratory and of the research laboratory. The positive Wassermann reaction at this stage of the disease was not conclusive. Yaws was one of the diseases which gave a so-called paradoxic positive Wassermann reaction; that is, a positive Wassermann reaction in the absence of syphilis. It had been held, though, that if the positive Wassermann test was converted to negative with less treatment than was ordinarily necessary to convert the positive Wassermann test in a syphilitic patient to negative, the weight of evidence in a suspected case would lean toward the diagnosis of yaws rather than syphilis. Dr. Goodman would not advise dependence on such a procedure.

DR. THORNLEY said that the patient came from the tropics, but not all patients that come from the tropics have yaws any more than all patients from elsewhere have syphilis. The examination was made by the pathologist of the Marine Hospital, but there was no effort or attempt to differentiate between the dark-field picture of syphilis and of yaws. The interesting part of the case was that while during the past three years Dr. Thornley had seen many cases of syphilis at the Marine Hospital, he had seen only one other that was at all like this; both patients had papules on the face, with a tendency to vegetation. This man certainly had a roseola as noticed here, and as also mentioned in

the history; it was more easily seen in the day than at night. It was also a fact that he had an indurated scar on the penis, probably the remains of a chancre, but there was nothing in the mouth or fauces to suggest mucous patches. The Wassermann reaction was positive. Dr. Thornley said that he was willing to admit, and in fact believed, that the man had syphilis; but the fact that they had another case from South America almost exactly similar to this one made him think of the possibility of its being yaws.

NEVUS VASCULARIS (KERATOTIC TYPE). Presented by DR. ABRAMOWITZ.

E. B., a girl, aged 4, of Jewish parents, was born in this country. There was an ordinary flat angioma about three-fourths inch (1.8 cm.) in size on the right mid-clavicular region. On the inner side of the right calf were two raised warty lesions, the edges of which were still angiomatous. The lesions were about one half inch (1.2 cm.) in diameter, connected by a small bridge of warty nature, and had been present since birth. The father said that frequent bleeding occurred from the lesion on the calf.

LICHEN PLANUS HYPERTROPHICUS. Presented by DR. MITCHELL.

H. S., an Irish laborer, aged 73, married, six months before presentation noticed a reddish eruption on the body and shoulders, which itched intensely. The itching and eruption had grown worse ever since. He was first seen on Jan. 5, 1924, at which time he showed a reddish purple eruption on the body, which was made up of rounded scaling papules, many of which had coalesced. There were no lesions in the mouth and genitals. Since then he had had one papule on the shaft of the penis, and at the present time there was a grayish reticulated appearance of the mucous membrane of both cheeks. The biopsy showed lichen planus. The Wassermann test was negative. In the past two weeks some of the lesions had flattened, leaving areas of pigmentation. Most of the others had decreased in thickness. The case was shown because of the unusual distribution of lesions of hypertrophic lichen planus.

NODULAR LESION OF TONGUE. Presented by DR. ROTHWELL.

R. B., a woman, aged 38, married, born in the United States, presented in the middle of the dorsum of the tongue a red, practically noninfiltrated lesion, made up apparently of two lesions bilaterally placed on each side of the median line of the tongue. The condition was of one year's duration. There were no subjective symptoms. The Wassermann test was negative.

MULTIPLE EPITHELIOMA. Presented by DR. TRIMBLE.

A woman, aged 57, married, born in London, England, whose father and mother died at the ages of 74 and 73 respectively of unknown cause; on examination presented on the skin of the back about the region of the fifth dorsal vertebra an irregularly shaped, circular, scaly lesion of a reddish purple color, measuring one and one-half inches in its transverse diameter. The lesion was not elevated above the surrounding skin; the border was well definite, but did not have the "rolled" and "pearly" appearance usually seen in basal cell epithelioma. The lesion had appeared five years previously, but the patient could not state the length of time it took to develop to its present size.

MOLLUSCUM FIBROSUM. Presented by DR. TRIMBLE.

A woman, aged 33, born in the United States, presented of small tumors and pigmented lesions scattered over the body, mainly on the face, chest and back. The case was shown chiefly on account of the family tendency to this condition. The patient's mother had been shown previously and was practically covered with small tumors. The lesions began when the patient was 16 years old.

BASAL CELL EPITHELIOMA (PAGET'S DISEASE). Presented by DR. TRIMBLE.

A woman, aged 64, married, born in Scotland, whose family and previous history were of no significance, forty years before had noticed a denuded, weeping area of skin about one inch below her navel in the midabdominal line, about the size of a dime. There were no subjective symptoms. The lesion increased slowly in size over a period of many years, drying and crusting at intervals after the use of protective powders. Twelve years before, at which time it had attained a diameter of about 3 inches (7.6 cm.) it was irradiated in Tacoma, Washington, with eight or ten fractional doses until the production of a mild second degree reaction. This apparently had little effect. For the last six years the lesion had spread rapidly. The microscopic section proved the lesion to be one of basal cell epithelioma, and was strikingly similar to the pathologic picture of Paget's disease.

URTICARIA PIGMENTOSA. Presented by DR. LEVIN.

L. H., a man, aged 44, a waiter, an Austrian by birth who had been in this country for about thirty-five years, reported that he had noticed the eruption for which he sought relief for about three years. He had always had freckles, but recognized that the eruption of three years' duration was not the same. The eruption was spreading, and at the time of examination covered the extremities and trunk. He presented several types of lesions: (1) verrucous lesions on the backs of the hands; (2) numerous small fleshy moles, nonpigmented, on the neck and shoulders; (3) pigmented lesions of lentigo; (4) pinhead to pea-sized red to violaceous raised areas on the arms, legs and trunk, which did not fade out entirely on pressure, but left areas of pigmentation; (5) telangiectatic lesions, especially on the flanks, with apparent circinate outline, but these lesions and the other raised spots described above did not have any orderly arrangement one with the other; (6) frank dilated capillaries on the lower extremities, and several such in the middle of the back; (7) an orderly arrangement of bluish macules which faded on pressure over the internal malleoli, and along the inner aspects of the soles. The patient was married, had three living children, and reported no miscarriages. The blood Wassermann test was negative. Blood chemistry revealed: urea nitrogen, 16.2; normal, 15; nonprotein nitrogen, 37.7; normal, 35; creatin, 1.1; normal, 1; glucose, 0.125; normal, 0.120.

MACULAR ATROPHY IN A SYPHILITIC PERSON. Presented by DR. WALZER for DR. LAPOWSKI.

A. L., a woman, aged 63, was a widow, whose husband first came to the Good Samaritan Dispensary nineteen years before, with a history of ulcus durum thirteen years previously. He had suspicious syphilitic manifestations. He was treated for syphilis for five years, but his attendance was very poor

He died of heart trouble (probably syphilitic) the next year (nineteen years after the initial lesion, or six years after first appearance at the dispensary). His wife first appeared at the dispensary eighteen years ago, or fourteen years after husband was infected, with leukoderma of the neck and a papular syphilid over the body. She had six living children and no miscarriages. She also practically received no treatment on account of poor attendance. Eight years ago (ten years after the first visit) she returned with leukoderma of the neck and atrophic spots on the body. Last November she returned again with manifestations like the present ones. Over the entire body were scattered atrophic spots, pea to finger nail size, some of them (over the neck posteriorly) looking like leukoderma. Over the rest of the body were white atrophic lesions, with the skin over them wrinkled, raised, thinned and with no areola. They felt like holes covered with thinned skin and were arranged in no definite grouping. Sebaceous and sweat orifices were seen on the surface of the lesions. On the right side of the forehead, temporal region, was one red, infiltrated papule beside an atrophic spot. There was marked dermographism. Physical examination revealed an enlarged liver, two fingers below the costal margin—not tender, and smooth. The Wassermann test was negative.

Monilethrix. Presented by Dr. Walzer for Dr. Lapowski.

S. P., a girl, aged 9, was the oldest of three children, all healthy. The other two were not similarly affected. The present condition developed when the patient was 6 months, following an attack of "sickness." The hair had never grown properly since. At present the whole scalp was affected, the hair irregular in size from ¼ to 1½ inches, lusterless, easily broken, twisted or curved, and easily extracted from the follicles. The largest hairs were apparently normal. With a hand lens or naked eye, the characteristic "heads" and "constricted parts" could be seen in regular succession on the short hairs. There was keratosis pilaris involving the entire scalp. The tiny papules were red, slightly raised and each pierced by a hair. The flexor surfaces of the arms and outer part of the thighs were covered with numerous pin-point to pinhead pinkish or red slightly raised papules, nutmeg grater-like to the touch—keratosis pilaris. There was incomplete alopecia of the eyebrows, with hairs of different size. The hair of the eyelashes was of irregular size. The lanugo hair on the rest of the body was not involved. There were no other abnormalities. The teeth and nails were normal.

Nevus Pigmentosus et Pilosus, Bathing Suit Type. Presented by Dr. Wise.

R. M., a girl, aged 19, born in the United States, had a condition that had been present since birth. She presented the bathing suit type of pigmented hairy nevus, involving the trunk and thighs down to the knees, and up as far as the second dorsal spine. On the back of the hips was one nevus the size of a coconut. On the right hip, there was an extensive lipoma with olive-sized lipomas beneath. On the left side of the hip, there was a large orange size lipoma. On the face, trunk, and extremities, there were numerous pigmented nevi of varying size. There were some keloids on the chest resulting from application of acid. There were numerous pits on the face.

Dermatitis Medicamentosa (Phenolphthalein). Presented by Dr. Scheer.

A. R., a woman, aged 60, had had the condition for two years, with attacks every four or five weeks. It was located on the dorsal surfaces of the hands

and left wrist. She presented noninfiltrated, violaceous plaques from 1 to 2 inches in diameter, with moderate itching. There was a history of the ingestion of Ex-Lax.

PAPULONECROTIC TUBERCULID. Presented by DR. CHARGIN.

S. F., a woman, aged 33, housewife, the mother of two healthy children, had a history that was negative, except for an attack of "rheumatism" seven years previously, which kept her in bed for a period of several weeks. The present eruption began about nine months ago, the first lesion appearing on the face. Since then lesions had appeared all over the body, especially on the forehead, cheeks, arms, buttocks and legs. The largest lesions were observed on the legs. The most recent eruption was of six weeks' duration, and was located on the arms and legs. There were a great many typical scars scattered over the arms, face, and legs, and active lesions might be seen, especially on the forearms on the posterior aspect. There were also several other lesions on the face. The patient's general health was good. There were no enlarged glands, nor were there scars of former broken down glands. The Wassermann test was negative.

OSCAR L. LEVIN, Secretary.

ST. LOUIS DERMATOLOGICAL SOCIETY

Regular Meeting, Feb. 13, 1924

DR. W. H. MOOK, M.D., *Presiding*

A CASE OF CONGENITAL ICHTHYOSIFORM ERYTHRODERMA. Presented by DR. W. H. MOOK.

G. J. W., aged 22, had been observed by Dr. Mook three years previously, at which time he had skin lesions with a duration of nine years. On his arms, forearms, thighs, buttocks and legs were brownish-red, scaly patches from 4 to 5 inches (10 to 12 cm.) in diameter. They were fairly sharply defined, resembling inflamed patches of localized ichthyosis. They were absolutely symmetrical. The arm lesions were smaller, scaly and looked like old seborrheic dermatitis, distributed on the inner surfaces only, from axilla to wrist. There were a few on the chest. The culture for epidermophytosis was negative. The pigmentations were dark brown in the oldest lesions. On Feb. 5, 1924, the patient returned to Dr. Mook, the lesions at this time having the same distribution, but being more marked, the pigmentation also being deeper. The only new lesion at presentation was one on the outer axilla. (The case will be published in full in the near future.)

DISCUSSION

DR. W. D. DAVIS said that the lesions impressed him as being those of ichthyosiform erythroderma, similar to the lesions that appeared in a young girl presented by Dr. Weiss some time ago.

DR. GRINDON inquired whether there had been bullae in the case presented.

DR. MOOK replied that there had been none in this instance.

Dr. Weiss said that he thought this was a case of ichthyosiform erythroderma. In this connection, he mentioned a case that he had presented some time since which was of the so-called "bullous type," of which he and Dr. Tobias had been making a special study, especially in regard to the other features that accompany these cases.

Dr. Stryker said he thought the case resembled the seborrheic type of parakeratosis.

Dr. Kring agreed with Dr. Weiss' diagnosis, except for the lesions on the back, which he thought were acne vulgaris.

Dr. Tobias stated that he had seen four cases of this type at Washington University Dispensary and at Barnard Hospital and had reviewed the literature on the subject. Most authors considered it not merely a form of ichthyosis, but a clinical entity. The Germans call it ichthyosis rubra. It seems that the disease is characterized by a congenital nature and a familial tendency, and the involvement of the skin of the face and flexures. There are two types of this disease, the dry and the wet (bullous). Some writers consider the bullous lesions as simply due to secondary infection. Nothing can be done in the way of treatment.

A Case for Diagnosis. Presented by Dr. Garold V. Stryker from St. Louis University Dispensary.

Mrs. T., aged 37, had entered the clinic, complaining of pityriasis capitis. Examination disclosed two lesions on the neck just below the lobe of the left ear, which the patient stated had been present without changing in size as long as she could remember. The lesions on presentation were pinkish or salmon red, slightly elevated, oval, about 1 cm. long and 5 mm. in their greatest width. Close inspection revealed that each patch was made up of many pinpoint to pinhead sized yellowish nodules of a firm consistency, having a translucent appearance, with a fine, capillary network running between and over the nodules. Puncture of several of the nodules had produced only a drop of serum, and blood.

DISCUSSION

Dr. W. D. Davis said he favored a diagnosis of nevus, resembling somewhat a unius lateris variety, although on close inspection some of the lesions resembled lymphangioma circumscriptum.

Dr. Weiss said he had classified the condition as a nevus, but not of the unius lateris group, because of the lack of the verrucous feature. Underneath the skin there were yellowish-looking nodules, as if the lesions contained inspissated sebaceous material. He considered the possibility also of lymphangioma circumscriptum.

Dr. Stryker said that each patch was made up of numerous small yellowish nodules of translucent appearance. Running over them and between them were numerous capillaries. On puncture of one of the nodules, only blood and serum exuded. There was no sebaceous material and no prolonged lymph drainage.

Dr. Grindon offered a diagnosis of nevus, but considered the possibility of a diagnosis of adenoma sebaceum of the nonsymmetrical type.

Dr. Kring concurred in Dr. Grindon's diagnosis, having abandoned his original diagnosis of molluscum contagiosum, because there was no oozing from the vesicles.

Dr. R. H. Davis and Dr. Mook said they thought the lesion was a nevus.

Dr. Tobias agreed with them, favoring diagnosis of the unius lateris type, accompanied by hypertrophy of the sebaceous glands.

A Case for Diagnosis. Presented by Dr. Joseph Grindon.

A white man, aged 48, a night watchman, a large, well-developed man, whose general health had always been good, first had skin lesions nineteen years before presentation. At that time, he had been handling spoiled canned tomatoes. There were itching and discharging vesicles about the backs and borders of the hands and between the fingers, which later formed large pustules. This condition continued about one year. The second attack occurred four years before presentation, chiefly on the forearms, which were in part raw and moist. The present attack began 6 months ago on the fronts of the wrists. These became thickly infiltrated and pruritic, with deep fissures. They were greatly improved and the pruritus much relieved by the use of a sulphur salve. About four months ago, the forearms and then the arms were involved. Two months ago, lesions appeared about the knees, thighs, hips and over the sacrum. At presentation, both wrists showed patches of large papules and easily broken vesicles on infiltrated bases. These were quite symmetrical. The vesicles at times ran together into red, raw, wet patches. On the arms and forearms, there was no symmetry of the lesions, the right arm alone being involved. Bands and patches of discrete lesions extended up to the axilla and down on the chest to the nipple. These presented a curious arrangement. They passed up the ulnar border of the forearm nearly to the flexure of the elbow, whence a narrower linear strip extended to above the flexure and joined a patch about 1 inch (2.5 cm.) in diameter. From this point, two linear bands passed to the axilla, the more anterior, narrow; the more posterior-internal, broad. In the axilla itself were a number of lesions. On the chest just in front of and a little above the axillary fold was another patch one-half inch (1.2 cm.) in diameter, from which a broad band passed down the side of the thorax nearly to the nipple. From the middle of the forearm up, many of the lesions were flat-topped, glistening papules; many, especially in certain groups, presenting angular borders. Interspersed among these were some minute vesicles. On the upper part of the inner aspect of the left arm were some dark, flat seborrheic warts. Large symmetrical patches over the hips and upper part of the buttocks were joined by lesions stretching across the sacrum. A small patch midway of the outer aspect of each thigh was also symmetrically placed. There was a patch anteriorly over each knee, from the tuberosity of the tibia to about 3 inches (7.6 cm.) above the upper border of the patella. Over the anterior aspect of each ankle a retrogressing patch extended to the dorsum of the foot. The lesions on the lower extremities presented a different appearance from those on the upper extremities, being composed of distinct groups of vesicles, for the most part ruptured, the appearance here being highly suggestive of dermatitis herpetiformis. The scalp showed abundant pityriasis. The glands and all skin surfaces not mentioned were free. In the oral mucosa there was a small milk-white patch a little in front of the right anterior pillar. A thin milky streak ran back from the right oral angle along the buccal surface. There was no similar lesion at the left angle. There was a minute white patch near the tip of the tongue. The patient had been a heavy smoker.

DISCUSSION

Dr. W. D. Davis said he thought it was planus of the linear type with evidences also of the vesicular type. The lesions about the wrists and knees were grouped like dermatitis herpetiformis, but the lesions on the arm and in the axilla showed linear formation. Running down the arm, they were papular, angular, shiny and flat.

Dr. R. S. Weiss also offered a diagnosis of lichen planus. In addition to that, he believed that there was dermatitis venenata from some external application used as a remedy for the pruritus. He described a similar case he had observed in a man, aged 55, with scattered lichen planus which was intensely pruritic. The man used a patent medicine to allay the pruritus, which produced areas of vesiculation. This patient had had an attack of lichen planus twenty years previously and had been treated by Dr. Ravogli and another dermatologist in the East, both of whom had given arsenic by injection. He was symptom-free for about twenty years. While in St. Louis, he had a recurrence which yielded to mercury and arsenic, but the man continued to resort to patent medicine locally for the pigment patches, which produced areas of vesicular dermatitis.

Dr. Grindon said he was puzzled as to a diagnosis. The lesions about the wrists and all the lesions from the waist down were strikingly symmetrical, while those on the right arm and chest were asymmetrical and resembled lichen planus of the linear type. As remarked by Dr. Weiss, the use of sulphur menthol might have something to do with the present appearance of the lesions on the wrists, although he hardly believed it was responsible for the lesions from the waist down, on account of their perfect symmetry. A striking point was the rapidity of the process. The lesions midway of the thighs two days ago were prominent, but it had rapidly diminished. Again, the follicular distribution of the lesions below the waist did not suggest lichen planus or dermatitis herpetiformis.

Dr. Conrad said he thought the lesions on the arm and chest were those of lichen planus, but those on the lower limbs looked more like lesions of the dermatitis herpetiformis group, which would change in time. He thought it a question as to the possibility of the patient having two separate and distinct skin conditions.

Dr. Mook diagnosed the case as dermatitis herpetiformis. The linear lesions resembling lichen planus on the arm were glistening, but with perhaps deep-seated small vesicles, and giving the appearance of a papular group. The fact that they left pigmented spots on the lower extremities with symmetrical arrangement in groups would suggest dermatitis herpetiformis.

Drs. Weiss and Grindon remarked that the patches in the mouth were not leukoplakic, but more like the white patches in planus.

Dr. Robert H. Davis asked whether any one had observed a case of linear dermatitis herpetiformis?

Dr. Greiner said there were two distinct conditions, linear lichen planus on the arms; and from the waist down, a type of staphylococcic dermatitis with a follicular grouping, such as is shown clearly at this presentation.

A Case of Paraffinoma. Presented by Dr. Richard S. Weiss.

A white woman, aged 54, fifteen years ago had paraffin injected under the skin of the forehead to remove wrinkles. The tumor began growing about

two years ago. At presentation a hard mass, about 4 by 3 cm., was observed on the forehead just above the nose. The skin above it was reddened in patches.

DISCUSSION

Dr. Mook said that until he had heard the history of the case he had felt the condition was like a calcified sebaceous cyst, being encapsulated, sharply defined, with no metastatic-like lesions in the subcutaneous tissue, such as are seen in a paraffinoma. Paraffin after a number of years usually begins to run, following the lines of the muscle fibers, when small new tumors will begin to form, like metastatic growths. In this connection he thought it of interest to mention a similar case, one now in the courts, in which a suit was brought against a drug firm because of alleged injury from the effects of injection of camphor in paraffin oil. In that case an attempt was made to excise a paraffinoma from the arm, excising it only partially. After the operation it began to form metastatic-like growths, like carcinomas, over the chest wall and down the arm. The operation had served merely to stimulate the formation of new growths which became larger than ever. Dr. Mook warned that it was well to remember that unless the paraffinoma can be excised in its entirety, it is wiser to leave it undisturbed, as operative procedure makes the growth spread like wildfire in many instances.

Dr. Mook replied that many cases had been reported in France, but there had been no malignancy. He mentioned in this connection that Dr. Montrose T. Burrows, director of laboratories at Barnard Free Skin and Cancer Hospital, had been experimenting in the production of malignant growth with paraffin oil in animals. He also mentioned that in one of his private cases the lesion had broken down, following pressure of the oil on the skin, as in a gumma, with, however, subsequent healing.

Dr. Weiss related how this patient, fifteen years previously, had gone to a physician who "guaranteed" to remove the wrinkles above her nose, which result was apparently obtained, the paraffin holding well until two years ago, at which time the tumor began to grow. He proposed to have the tumor removed surgically, en masse, and, if necessary, to do a plastic operation afterward.

A Case of Sensitization Dermatitis. Presented by Dr. W. D. Davis.

A white man, aged 56, married, a cigar salesman, gave a negative history, except for having pneumonia a few years previously. He said that an eruption had appeared on his hands and forearms in January, 1921, which had lasted until April, this condition having again made its appearance in January, 1922, lasting until April of the same year. On Dec. 28, 1923, the eruption had appeared a third time, growing progressively worse up to the time of presentation. Having been treated elsewhere without relief, he came to the outpatient department on February 9, with the following skin picture: The face, neck, trunk and extremities presented an acute erythematous, scaly, fissured, and in some places, a weeping and crusted dermatitis which was very pruritic. The eruption extended about to the ankles. The hands were swollen.

DISCUSSION

Dr. W. D. Davis said that the regularity with which the eruption had recurred was the sole reason for presenting the case, appearing as it had done, between January and April of each year. In his opinion it was a case of sensitization dermatitis probably due to the handling of tobacco.

Dr. Mook remarked that the etiologic factor in these cases was most important. Ten years ago, these cases were thought to be a sensitization dermatitis due to some internal cause, but in view of the study of more recent years, a great number of cases of symmetrical diffuse dermatitis over the body were known to be due to external causes. He described a case similar to that under observation, in which there was a well developed acute dermatitis all over the head, perfectly symmetrical, and involving all the follicles of the skin, due to paint and chemicals. It began on the toes, extending upward to the point where the sleeve protected the body, the arms and forearms being free. It was a diffuse acute dermatitis; in other words, it covered the areas of external irritation and spread by rubbing; i. e., sensitization due to an external cause. During the summer, there are many and various eruptions due to sensitization to plants with which patients come in contact. The present condition, Dr. Mook thought, was due to some unknown factor, some external agent with which the patient had come in contact during the winter months.

A Case for Diagnosis. Presented by Dr. A. H. Conrad from Dr. Engman's Clinic at Washington University Dispensary.

A white man, aged 45, a printer, had a skin disease that began on the right leg eight months previous to presentation, and on the left leg about six weeks previously. When first observed, he presented a patch of chronic scaly dermatitis, roughly oval, about 2 by 1½ inches (5 by 3.8 cm.) on the lower third of right leg, and a small patch about 1 inch (2.5 cm.) in diameter on the lower third of the left leg. He was given Whitfield's ointment and returned in a few days with the present condition, consisting of an oval lesion, 6 by 3½ inches (15 by 8.8 cm.) on the right leg, in the center of which was an irregular patchy area of dull red to brownish dermatitis. Outside of this was a ring of fairly normal skin, and outside of this, a peculiar limiting ring of dermatitis about one-quarter inch (0.6 cm.) wide, sharply demarked from the surrounding skin, the central portion being slightly depressed. There were no papules and no punctate hemorrhages.

DISCUSSION

Dr. Grindon said he did not think a diagnosis possible at this time after the application of Whitfield's ointment, which had materially changed the condition.

Dr. Kring agreed with Dr. Grindon.

Dr. Conrad said he could not believe that Whitfield's ointment could cause this dermatitis with a ring around the lesion, leaving perfectly healthy skin in the center. He inquired as to why the edges only were affected, and not the center.

Dr. Grindon replied that the condition seemed to confine itself to the site covered by the margin of the dressing where the ointment perhaps accumulated.

Dr. Mook said that he remembered a patient whom he had treated many years ago, who had a spot on each knee, scaly and red, the border only showing glistening, pigmented skin where the lesion had formerly been and had become smaller. He believed the remedy given, consisting of salicylic acid, chrysorobin and pyrogallic acid, 0.5 per cent. each, had been responsible for the condition noted. In the New York Skin and Cancer Hospital, this was used in stubborn cases affecting the elbows and knees, and within a week or two, exactly the same condition as shown in this patient had occurred. It produced a great ring, 4 inches (10 cm.) in diameter, within two or three months. Then followed

a general outbreak of psoriasis, and the patient had blamed him for the spreading of the lesions, which later proved to be psoriasis. Dr. Mook said he thought the case under observation was also psoriasis, because of the features noted; that is, the ring formation, the spreading of the lesions, etc.

A Case of Lichen Planus of the Trunk and Mouth. Presented by Dr. Norman Tobias from Dr. Engman's Clinic at Washington University Dispensary.

A white man, aged 49, had an eruption which began four years before, which was mildly pruritic at times. The eruption was on the trunk, chiefly in the seborrheic areas, consisting of plane, flat-topped papules in small plaques, and many single dull red lesions with central depressions. There were typical white mosaic patches on the mucous membrane of the cheeks.

Dr. Greiner said he thought it was a case of lichen planus, being at first puzzled in regard to a diagnosis until he had observed the lesions in the mouth which were typical planus lesions.

Dr. W. D. Davis agreed with Dr. Greiner that the case presented an atypical clinical picture of lichen planus, with the peculiarity that the lesions were confluent over the seborrheic areas of the chest and back, and more discrete over the sides of the chest. He did not think the lesions themselves were typical, many of them not having the characteristic angular papules of lichen planus; many of them, on the other hand, were violaceous, with shiny flat tops. The lesions in the mouth were typical.

Dr. Weiss said he thought it was a typical case of lichen planus. The plaques in the mouth were especially typical.

Dr. Kring said he considered it an atypical case of lichen planus, since in all the cases he had observed the lesions were not in the seborrheic areas, but often on the lower limbs and frequently on the flexor surfaces of the arms.

Dr. Tobias said that this case was of special interest, as it served to show how frequently a case of lichen planus may resemble syphilis, especially with the lesions in the mouth.

CHICAGO DERMATOLOGICAL SOCIETY

Regular Meeting, Feb. 20, 1924

James Herbert Mitchell, M.D., *Presiding*

Parapsoriasis. Presented by Dr. Senear.

The patient had had a generalized eruption for the last twenty-one years, with the history that it had been worse during the winter months. She presented a parapsoriasis of the small papular type. As she was greatly overweight and had a slow pulse, she was given thyroid medication, and after ten days pituitary gland was added. In two months she had shown remarkable improvement, and was enthusiastic about the result, particularly at this time of the year. No local medication was employed.

Dr. Senear said he was not drawing any conclusions as to the permanency of the effect of the treatment or the possibility of the condition clearing up entirely. The patient said it was the first time she had been able to spend a comfortable winter. She had lost only one-half pound in weight. She had received one-third grain (0.02 gm.) of thyroid three times a day to start with,

then one-third grain twice a day when the pituitary gland was added. The thyroid had been increased for the past ten days and she was receiving 1¼ grains (0.08 gm.) daily.

DISCUSSION

DR. STILLIANS said he was glad to know the patient had made some improvement and hoped there would be a further report on the case.

DR. RAVITCH asked whether it was the thyroid or the pituitary gland that was producing the improvement.

ACNITIS. Presented by DR. SENEAR.

This patient gave a history of an eruption which began four months before presentation, appearing as a typical sycosis barbae of the upper lip. He saw Dr. Charles Collins, who gave him some violet ray treatment. Dr. Senear first saw him three months before. The condition on the upper lip involuted readily, leaving a verrucous surface. The eruption on the face had varied greatly from day to day. At times the lesions were small, and again they were deep-seated, conforming to acne lesions. About four weeks before, he developed lesions on the back of the hand. These were deep-seated, and in the process of evolution had developed into lesions similar to those on the upper lip. He had also developed a number of lesions on the back of the neck, some of which were in the hairline. These seemed to be the same as the deep-seated lesions on the face.

DR. SENEAR said the case had impressed him very much like a case of active acnitis shown by Dr. Quinn some three or four years ago.

The lesions on the upper lip seemed at the start to be a definite sycosis barbae. He showed lesions of the same type on the knees.

GRANULOMA FUNGOIDES. Presented by DR. GRANT (by invitation).

A widow, aged 54, of French descent, ten years before, first noticed sharply circumscribed red spots about the left knee. These were replaced by others as they disappeared and were accompanied by itching. A year later similar spots appeared on the right and then on the left arm. There were no further lesions until about one year before, when erythematous lesions appeared on the flexor surfaces of the forearms, also associated with itching. Following trauma from broken glasses in September, 1923, the face became red, and small tumor-like lesions appeared, accompanied by intense itching which was worse at night. Various patent remedies were used, but the eruption spread to the trunk and extremities. The family history was negative.

The lesions began as red spots, and fungoid masses developed later. They were first red, then itchy, then serous, followed by the formation of crusts and tumor masses. Some lesions had contained pus and some had resolved spontaneously. The lesions had frequently followed trauma, and there was a history of the lips becoming swollen at times, the swelling rapidly disappearing. Considerable itching had been present after eating strawberries and cranberries.

Examination revealed fungoid lesions, sharply circumscribed, elevated and crusted, dull red, erythematous, infiltrated and scaly, scattered over the left side of the forehead, cheeks, neck, abdomen and extremities.

The blood Wassermann reaction was negative, and the urine was negative for albumin and sugar.

DISCUSSION

DR. OLIVER said he thought it was a case of mycosis fungoides.

DR. STILLIANS said it was unusual to see a case of this kind develop with such rapidity.

SCLERODERMA. Presented by DR. OLIVER.

A boy, aged 13, presented a scleroderma involving entire right half of body. The condition was of one year's duration, and began with pains and stiffness in the joints.

DISCUSSION

DR. PUSEY said he thought it was a case of scleroderma with atrophy.

DR. LIEBERTHAL concurred in the diagnosis, but said the rapid course of the case was rather surprising, as atrophy did not usually take place so fast, especially in such young patients. In these the course was, as a rule, more favorable.

DR. OLIVER said he was very much interested in the neurologic aspect of the case. This had not been worked up as yet, but the case was to be thoroughly studied from this standpoint. He thought it agreed in some ways with the case reported recently by Williams in the ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY. Two neurologists had seen the case. One thought there was a lesion in the posterior root ganglion, but the other had not given a definite opinion.

A CASE FOR DIAGNOSIS. Presented by DR. ZEISLER.

A boy, aged 10, had had a peculiar condition on the ears for four years. He had lived in the Orient, China and Japan until he was 4 years old, and since then he had lived in California. This condition started two years after he went to California. He was presented at the request of Dr. J. Gordon Wilson.

When Dr. Zeisler first saw him he had an ulcer on the border of the ear, which he had at first suspected of being an "oriental sore." Smears for organisms, however, were negative. The patient later informed him that a physician in Quincy, Ill., some time before had removed a section of the ear. There were present in addition some small, grouped, firm, yellowish cysts and pitted scars. The other ear showed some scarlike areas.

DISCUSSION

DR. PUSEY said he thought it was a congenital defect of the skin involving the sebaceous glands. The ear was rather larger than a normal ear and even larger than the other ear. It had some of the characteristics of the flabby areas he had seen in unmistakable nevi of the auricle. There was a little of the condition on the other ear, but not much.

DR. RAVITCH asked whether the boy could have developed disturbances of the circulation of the glands.

DR. FOERSTER said he thought of the possibility of the condition being related to the atrophy which was described as occurring on the cheeks. This condition had been described by MacKee and others. He thought also that it might be acne.

DR. ZEISLER said that Dr. Wilson had attributed the condition to some infection the boy had acquired in the Orient. That was why he thought it might be an Oriental sore. Later he learned that a piece had been removed from the ear a short time ago, which might account for the ulcer.

LYMPHANGIOMA CIRCUMSCRIPTA. Presented by DR. MITCHELL.

This case was demonstrated last spring and was presented at this time to show the effect of treatment. The patient had received mild radiotherapy since last spring and had improved greatly.

LUPUS HYPERTROPHICUS. Presented by DR. STILLIANS.

A negress, a widow, aged 34, a native of Louisiana, complained of sores on the nose and upper lip. The lesions first appeared seven years before, following an injury to the left ring finger. The patient first noticed a stinging sensation which was soon followed by pain, swelling and the formation of a nodule. Operation gave no relief, and the infection spread to the little finger and then to the nose. A growth developed in the nose, which was operated on in 1916, and following operation a papule appeared on the nose, which gradually spread and formed the lesion now present. The right thumb became involved in 1918, followed by gradual swelling of the arm and involvement of the right index finger. About three years before she had first noticed a small warty growth on the great toe.

Covering the entire nose, upper lip and inner half of both cheeks there were numerous discrete and grouped soft, reddish-yellow and reddish-brown nodules. The right forearm was elephantiasic in appearance, with scattered brownish nodules and papules. The toes showed a verrucous type of lesion.

DISCUSSION

DR. PUSEY said he thought the lesion on the face was lupus and that the other lesions were tuberculosis of the skin.

DR. OLIVER said that the sections showed tuberculosis, and roentgen-ray examination revealed definite bony changes in the hands. He did not think the condition belonged to the sarcoid group.

A CASE FOR DIAGNOSIS (SCHAMBERG'S DISEASE?). Presented by DR. BEESON.

A man, aged 22, said that the trouble began about three years before, first on one leg and then on the other, as little reddish points. There was no history of any other skin disease. The past history was negative except for whooping cough at the age of 2. His Wassermann reaction was negative.

DISCUSSION

DR. ZEISLER said he thought the condition was traumatic.

DR. OLIVER said he did not know what it was. He did not believe it was Schamberg's diseases.

DR. MCEWEN said he would consider the possibility of a dermatitis medicamentosa.

DR. PUSEY said he thought it was a typical case of follicular hemorrhage followed by pigmentation in the skin of the legs. If a name was necessary, it could be called dermatitis hypostatica. When any one had an injury to

the capillary blood vessels that was followed by extravasation, these pigmentary deposits were the result.

Dr. Stillians said he believed there was something more. There must be some associated susceptibility; the blood vessels must be especially susceptible to injury. Ordinarily such pigmentation did not occur when a person knocked his chin.

Dr. Beeson said he had presented the case to ascertain whether any one would think it was related to Schamberg's disease. He believed there was an element of trauma and some other factor. He was going to make sections to find out whether there was an inflammatory element, and he wanted to know whether any one could tell him what to use to cure the condition.

Dr. Pusey advised the use of a sulpho-ichthyolate preparation and collodion dressing. This does not wash off, relieves the itching, and can be applied as often as necessary.

A Case for Diagnosis. Presented by Drs. Senear and Cohen.

A negro, aged 29, who gave a history of having had a tender bubo in the inguinal region last November, early in January developed a follicular eruption. His Wassermann reaction was + + + +. He had had no lesion on the penis previous to this attack and had none at the time of presentation. The blood serum had never been examined before, so there was nothing known about his past history regarding syphilis. There was also a pigmented nevus in the region of the left breast and an eruption over the flanks.

DISCUSSION

Dr. Zeisler complimented Dr. Senear on detecting these cases of lichen spinulosus.

Dr. Senear said he was interested in this case not so much from the standpoint of the clinical type of the lesion found in lichen spinulosus, but in this group of diseases generally. Several cases of this type had been shown here and at the St. Louis Dermatological Society meeting, and they were all in colored people. He believed there was something in the colored skin which made it particularly subject to this type of disorder.

A Case for Diagnosis. Presented by Dr. Stillians.

An Italian, a cook, presented himself on February 2, complaining of an eruption on the genitalia and on the hands, which had been present for the preceding month. The eruption consisted of slightly elevated, deep red plaques, which were somewhat scaly. These plaques were also present in the vicinity of the buttocks. On the hands, the eruption covered nearly the whole palm and the palmar surface of the fingers. There were a few lesions on the scalp. The Wassermann reaction was positive.

DISCUSSION

Dr. Eisenstaedt said he thought it was a case of psoriasis with an underlying complication. He thought the individual lesions were quite typical, but was uncertain as to the associated syphilis.

Dr. Zeisler said he thought the patient had both syphilis and psoriasis. Two Wassermann reactions were anticomplementary.

Dr. Mitchell said that from time to time cases were seen in the clinic with psoriasis in which the lesions became chronic on the hands as a result of the character of work done by the patients. He recalled a man who worked in a vinegar factory and had his hands in brine all the time, who had an active psoriasis on the palmar and dorsal surfaces of the hands. The process was much more active than in the patient presented.

Dr. Stillians said that since the patient was a cook possibly the occupation had something to do with localizing the eruption in the folds of the body and on the hands. He would try to determine whether the palmar lesions were syphilitic. Potassium iodid and mercury would not be likely to clear up psoriasis.

Dr. Zeisler said he had been given arsphenamin, and the palmar lesions showed distinct improvement.

Dr. Oliver said that one thing against syphilis of the palms was the fact that there was considerable involvement of the dorsal surfaces of the fingers. He saw the patient the first day he came into the County Hospital. He showed a typical psoriasis on the scalp and knees with this inverse type on the palms and backs of the hands. He did not think it was syphilis.

Dr. Stillians said he thought it should be considered a psoriasis until it was proved otherwise.

Dr. McEwen asked why one could not add to the diagnosis dermatitis from handling sugar. The patient complained of itching of the hands; why might not the condition be influenced in the direction of a dermatitis from his occupational contacts?

Dr. Foerster asked whether the blood sugar had been estimated.

Dr. Stillians said it had not.

A Case for Diagnosis. Presented by Dr. Eisenstaedt.

A man, aged 40, a tailor, for the past fourteen months had been developing nodules which came with some discomfort and actual pain, and then disappeared. They appeared on the arms and had been more active than at the time of presentation, although some active ones were present on the extremities. All the lesions had passed through the painful stage. There had been no nodules on the face. There was no history of tuberculosis in the family; the Wassermann reaction was negative, and the blood smear showed no changes. The patient had not been sick, although at the time of presentation he felt weak.

DISCUSSION

Dr. McEwen said he was not sure what the condition was, but he thought it had something of the appearance of an erythema nodosum. In seeking to establish a diagnosis, he would investigate for a possible focus of infection. The man looked ill, complained of shortness of breath, and it seemed to him probable that he was the victim of a prolonged infection with some form of low grade organism.

Dr. Pusey agreed with Dr. McEwen. He further believed that the man was ill, and that he had a general septicemia which was producing the infarcts.

Dr. Stillians said he thought it was a sarcoid or tuberculid of the erythema induratum type, and believed this diagnosis would agree with what Dr. McEwen had brought out.

Dr. Eisenstaedt said he brought the patient in for a diagnosis of his condition. His point of view was somewhat like that of Dr. Stillians, and he believed the case could be classed with the group of disseminated nodular sarcoid. Many of them are generalized. This man had been working all the time and had been sent to the medical clinic for some pathologic condition, the nature of which Dr. Eisenstaedt could not recall. He would make efforts to have blood cultures made and to ascertain other foci of infection. The condition had been present since Christmas, 1922, but the patient's ability to work had not been reduced. There was no history of a rise in temperature.

Dr. Pusey said that the lesions came and went in two weeks, according to the patient's description. He had prejudiced himself against the diagnosis of sarcoid, and the taking of infarcts of the skin and putting them into the sarcoids did not appeal to him as clarifying anything. The man looked sick and had told him that he did not feel like working and had not been able to do so for a long time.

Dr. Eisenstaedt said he would employ the necessary laboratory methods and endeavor to find out what the trouble was.

Dr. McEwen said that Dr. Ebert had made sections from the case of multiple, benign, tumor-like new growths, which was shown at the January meeting. The patient had been seen twice since, and a well defined nephritis had been discovered. Dr. McEwen expected to see the man again and to make a more complete physical examination.

Dr. Senear reported on the case of the Jewish woman with the bullous eruption, which was presented at the January meeting. The weak ammoniated ointment was continued, and the lesions had completely cleared up. At the time of presentation the case looked like a simple impetiginous process, but Dr. Wein told him that the patient had just developed the typical picture of pemphigus.

MINNESOTA DERMATOLOGICAL SOCIETY

Regular Meeting, Feb. 6, 1924

H. G. Irvine, M.D., *Presiding*

Leukoplakia. Presented by Drs. Irvine and Turnacliff.

The patient with leukoplakia was presented at a previous meeting, at which time there was a question as to whether the lesion was carcinomatous. Microscopic examination was negative. The patient was presented to show the result of treatment.

DISCUSSION

Dr. Sweitzer recalled a case he showed at the summer meeting, in which he came nearly missing the carcinoma, it was so near the edge of the lesion. That man is still alive after cautery and radium treatment.

Dr. Irvine stated that this man was given just the same treatment as if the lesion had been carcinoma. The patient was shown at this time to demonstrate the result of treatment with radium and cautery.

Syphilis, Secondary. Presented by Dr. Turnacliff.

A woman, aged 22, presented a generalized papular eruption from pin-head to pea size, some of which on the forearms resembled a lichen planus, while

the lesions over the small of the back and the abdomen presented a scaling suggestive of psoriasis.

DISCUSSION

DR. IRVINE said he thought it was too bad that cases like this are not demonstrated before the County Society so that the men in general practice could see them.

A CASE FOR DIAGNOSIS. Presented by DR. ODLAND.

A man, aged 36, a baker, had had recurrent attacks of an erythematosquamous dermatitis on the hands for twenty years. He had a marked hyperkeratosis of the plantar surfaces and sodden scaling between the toes. *Epidermophyton inguinale* was found in the tissue scrapings of the plantar surfaces or the interdigital spaces of the toes. No fungi were demonstrated in the scrapings from the hands. The case was presented for diagnosis of the condition involving the hands.

DISCUSSION

DR. ODLAND thought that this patient had been seen by half the members of this Society, and he did not know that any one had made a diagnosis. He was inclined to think that it was a case of epidermophytosis of the hands and feet.

DR. IRVINE asked what treatment the patient had had.

DR. ODLAND stated that he thought the patient had been treated only for eczema. He had been observed by many of the leading dermatologists of Europe as well as this country.

DR. SWEITZER stated that, if he remembered rightly, one man said the lesion on this patient's hands disappeared when he quit work. That would make one think of an occupational disease.

DERMATITIS HERPETIFORMIS. Presented by DR. TURNACLIFF.

A man, aged 34, has had itching grouped vesicular lesions over the shoulder blades, buttocks, backs of thighs, and over the ilium for two years, leaving pigmentation and scarring. The patient had received violet ray to the point of reaction weekly up to three weeks ago and the skin was about clear then, but now presented a new crop of rather large vesicles, one about the size of a pea.

MONILETHRIX. Presented by DR. SWEITZER.

The patient, a girl, aged 14, presented herself for falling hair. The scalp showed thinning of hair, over the top especially. Individual hairs showed the fusiform swellings separated by the constricted, atrophic portions. The patient stated that the condition followed an attack of chorea.

DISCUSSION

DR. FELDEN stated that he had seen one case of monilethrix or aplasia moniliformis in a child, aged 8 years, and the striking characteristic of that case was that the scalp looked entirely bald on the affected parts similar to microsporia, limited to the occipital region, but closer inspection through the magnifying glass showed that there were short hairs not longer than from 0.5 to

1 cm. which were alternately and regularly constricted and swollen. The follicles presented a very marked keratosis pilaris with acuminate elevations, while the case here presented only long hairs with alternation of pigment, white and black parts, which caused the seeming change of shape. There was a certain scantiness of hair but no short hairs. He would consider this case to be one of pili annulati, which means alternating pigmentation, white and colored segments in single hairs, belonging to the group leukotrichia.

Dr. Odland asked if there was not usually a loss of hair.

Dr. Felden stated that in cases of pili annulati, there is not usually a loss of hair, but the fact that this patient did not have a large amount of hair could just as well be due to some other constitutional disease. It did not show the characteristic features of monilethrix.

Dr. Sweitzer stated that he thought Dr. Felden's examination was not as careful as his made under sunlight. Then it appeared that the entire upper part of the head had suffered from a considerable loss of hair. There is a lot of baldness on the back of the scalp. Her hair is very thick in places. He stated that some of the books class these as the same condition. This child did not have any hyperkeratosis, and apparently it is not in every hair. She did have this alopecia and claimed it came after chorea.

(Note.—A later examination showed the irregularities of monilethrix and pili annulati both present.)

Syringocystoma. Presented by Dr. Sweitzer.

Mrs. M., aged 31, presented numerous small yellowish soft tumors on the face, body and arms. The lesions have been present for the past ten years.

DISCUSSION

Dr. Sweitzer stated that this patient came into the clinic, and they had considered the possibility of an urticaria pigmentosa and also considered multiple benign cystic epithelioma, on account of the face being so widely involved. However, the more he saw of her the more he thought she had syringocystoma and biopsy showed syringocystoma on the forehead and neck, and there were also lesions under the eyelids. He stated that the patient said this had been coming on for about ten years. Some of the lesions are darkly pigmented, and that is the reason they had thought of urticaria pigmentosa.

Dr. Michelson said one of the most interesting things to him in this type of lesion was that the lesions looked fairly large, but when one examined the microscopic section, instead of finding cysts that corresponded to the size of the lesions, one found a collection of minute cysts.

Circinate Lesions on Trunk. For Diagnosis. Presented by Dr. Freeman.

The patient was seen for the first time about four days ago. The lesions were exactly the same as at present. He does not know how long the lesions have existed, as he had consumed considerable alcoholic beverage and slept in his clothes most of the time. The lesions are scattered over the trunk; and there are a few on the legs and neck. They are distinctly circular on the chest and oval on the back, and seem to follow lines of cleavage of the skin. They are erythematous and disappear on pressure. He has not taken any medicine of any kind for two weeks. The Wassermann was negative. The case was presented for diagnosis.

DISCUSSION

Dr. Armstrong thought this was a rather unusual case of pityriasis rosea. He stated that he had never seen one quite like it himself. In connection with pityriasis rosea, he mentioned the fact that he had seen two patients recently, a man and his wife, with this disease. That was the first time he had ever seen cases with the possibility of infection from one person to another. The duration between the outbreaks was about six weeks.

Dr. Sweitzer stated that in making a diagnosis, the following must be considered: syphilis, a toxic condition and pityriasis rosea gigantica; it looked to him more like pityriasis rosea gigantica, with reservations probably.

Dr. Irvine stated in regard to the point which Dr. Armstrong brought up, of infection from one to another in pityriasis rosea, that Mrs. Irvine once had pityriasis rosea and no precautions were taken to prevent other members of the family becoming infected, but no one was affected. Dr. Irvine felt that a diagnosis of syphilis was not easily ruled out in this case. The fact that the man had consumed a large amount of alcohol might have something to do with the negative Wassermann reaction.

Dr. Sweitzer said that he thought Dr. Michelson would remember a patient they had had at the hospital recently who looked as if she had syphilis of the face. The lesions had a very distinct border with no scaling of the center. They were inclined to make a diagnosis of syphilis, but on making a biopsy it proved to be lupus erythematosus, and the blood test was negative.

Dr. Butler thought this was a case of pityriasis rosea, as there was no scaling in the center area, and if one stood away from the patient it was salmon colored. He thought one need not hesitate about ruling out syphilis as it would only correspond to the circinate lesion of syphilis, and that could be ruled out by the fact that in early syphilis the patient would show concomitant signs of the disease. He had seen two cases of pityriasis rosea resembling this very much except that there are no vesicles in this patient.

Dr. Michelson said that he was unable to make a positive diagnosis, but he could not agree with the diagnosis of pityriasis rosea. He said that he could not raise peripherally attached scales from any lesion with a knife blade, and the lesions were distinctly palpable. He did not venture a diagnosis of syphilis, but he thought the course in the next two weeks should give conclusive evidence.

Dr. Butler said that there are two or three distinct types of pityriasis: the macular type, which shows scaling throughout the trouble, and which later shows scaling on the border, and then the true pityriasis rosea circinata. He thought this was the latter type.

Dr. Olson said he did not see how one could rule out seborrhea corporis, and he felt inclined to a diagnosis of an annular form of seborrhea corporis. The history of the case might suggest this.

Dr. Felden stated that there was no scaling and no infiltration. In syphilis cases with this type of circinate lesions, one would expect them to belong to the late secondary period and when we would not have such a large number of patches. In addition, the color does not show the dirty copper-brownish hue, characteristic of syphilis. Dr. Felden stated that his impression of this case is that it is a toxic dermatitis.

Dr. Cook stated that the fact that the man had not had his clothes off for two weeks might irritate a seborrhea corporis.

MORPHEA. Presented by DRS. IRVINE and TURNACLIFF.

M., a woman, aged 22, single, whose condition started ten years ago, had a lesion that began with small white spots which gradually grew larger and then became brownish. Some itching was present. At the time of presentation these several small white patches on the right shoulder were grouped, and with very slight pigmentation surrounding them. These were the latest to develop; the white skin was firm but not indurated. Over the abdomen and thighs were about a dozen more or less rounded or oval plaques from walnut to palm size consisting of mottled pigmentation in which there was apparent slight atrophy. Suggestions as to therapy were asked.

DISCUSSION

DR. MICHELSON said he thought this case was extraordinary, in that there was such an extreme amount of dark pigmentation surrounding the lesions.

DR. SWEITZER recalled seeing a case last summer which had pigmentation quite similar to this one. That was in a child.

DR. BUTLER said that the fact that in the lesions on the neck there was no pigment and there was some confluence, and decidedly white, almost ivory-like, would make him think of the so-called "white-spot" disease. Scleroderma is practically the same disease. He agreed with the diagnosis that it belongs to the scleroderma group.

INITIAL LESION OF LEFT TONSIL. IMPLANTATION OF RIGHT TONSILLAR FOSSA THROUGH TONSILLECTOMY. PRIMARY ADENOPATHY OF GLANDS OF NECK. SECONDARY SYPHILIS. Presented by DR. E. C. GAGER.

The patient, a man, aged 23, colored, a butcher, presented himself on Jan. 29, 1924. He had ulcerations in both tonsillar fossae. These ulcerations were superficial, covered with a grayish exudate and had sloping edges. There was extreme enlargement of the glands below the rami of both jaws. They were the size of goose eggs. There was a generalized papular syphilid with general adenopathy and a slight anemia. The Wassermann test of the serum was positive.

The patient gave the following history. One month ago he noticed that the left side of his neck and the left tonsil were swollen. He consulted a physician, who did a tonsillectomy. Thereafter the right side of the neck became swollen also. Three days ago the rash appeared.

The diagnosis in this case is, of course, presumptive, and not amenable to exact proof.

DISCUSSION

DR. ARMSTRONG thought this was an interesting case because of the question as to whether it was an implantation or secondary initial lesion from the scar on the tonsil on the other side. He thought it entirely possible, if we knew the exact time. He stated that he was under the impression that chancre can be reinoculated as late as seventeen days. He thought in this case we would have to give a nonproved verdict, as it could not be proved one way or the other.

DR. BUTLER thought there was no doubt that this man has syphilis and he believed the scaling skin eruption to be a syphilitic one. He thought the cervical adenopathy was something to consider, and to that end he believed

that a careful examination of the blood should be made, which should differentiate syphilis, leukemia, Hodgkin's disease, or, as many times following tonsillectomy, a two-sided sarcoma. He did not think those large glands were syphilitic.

Dr. Felden thought that the papular areas visible on the body were papular syphilitic lesions. He wished to know how Dr. Butler would bring that in connection with sarcoma, as a skin sarcoma could not show such an even distribution.

Dr. Butler thought Dr. Felden must have misunderstood him. He felt that the typical papular scaling was due to syphilis, but, in considering the glands of the neck it was necessary to differentiate from leukemia, Hodgkin's disease, etc.

Dr. Wethall thought the development of the glands in two weeks was rather rapid for sarcoma. He had known of cases to take two or three years.

Dr. Olson felt that the glands in the neck were rather large for syphilis, although they might be due to secondary infection. The mouth often harbors virulent bacteria. He was of the opinion that chancre in the mouth often shows a tendency to large infected glands.

Dr. Freeman said Dr. Gager stated the man had had these glands for only six weeks. That would rule out Hodgkin's disease or lymphosarcoma. He felt that the man had syphilis with secondary adenopathy.

Dr. Michelson said that all were agreed that the diagnosis was syphilis, but the course of events was a matter of personal interpretation. Dr. Michelson's interpretation was as follows: The man had had a chancre on one tonsil, and following the tonsillectomy, had developed an angina syphiliticum.

Dr. Sweitzer thought the probabilities were that the man had syphilis. He thought Dr. Butler's point was well taken from an experience he had had at the hospital. A young woman came in with a lesion that looked like Vincent's. It was treated but did not get better, and they thought she might have an initial lesion. In the meantime the glands on the opposite side persisted and kept growing. He had urged operation, and it proved to be lymphosarcoma. That was a rapidly growing disease, and it had fooled all of them for a while at least.

Dr. Irvine asked Dr. Gager to make a further report on this case at a later meeting.

Progressive Pigmentary Dermatosis. (Shamberg's Disease.) Presented by Dr. Michelson.

A youth, aged 19, presented on the anterior surface of the right lower leg scattered areas of a reddish brown pigmentation. The areas consisted of numerous pin-head sized pigmented puncta. The patient stated that the condition had begun several years before and had gradually enlarged. There were no subjective symptoms.

DISCUSSION

Dr. Michelson said he thought Majocchi's disease, angioma serpiginosum and Shamberg's disease were all closely related, and he could not see any distinct line of differentiation, especially between angioma serpiginosum and Shamberg's disease.

Dr. Goeckerman said he thought the diagnosis was correct, and he had little to add. He stated that if he had seen the patient first he thought he would have made the same diagnosis.

He also stated that they had seen a number of patients in the last two or three years, which cases they felt were difficult to classify. Following the description that Shamberg gives, it seemed that these would have to be classified as pigmentary dystrophy. It has been stated that Shamberg's pigmentary dystrophy was nothing more than an iron deposit in the skin formed as the result of venous stasis. Dr. Goeckerman did not believe that this was quite correct. He recalled a case where half a dozen patches were distributed all over different parts of the body. He thought we are justified in accepting the description that Shamberg gives as characteristic of the dermatosis.

Dr. Irvine recalled seeing several cases in Chicago. The patients presented had pigmented lesions on the legs. Two or three were left without any accepted diagnosis but were called Shamberg's disease, but they were not like this case at all. In one or two the pigmented areas were rather diffuse; there were no telangiectases. Among several of these doctors there seemed to be no fixed opinion of what Shamberg's disease really is. The cases referred to were not like this case at all.

Dr. Butler called attention to the fact that there was new pigment formation and a grouping of some of the central lesions into well-defined patches, and there were satellite lesions, especially around the lower leg. He believed that a diagnosis of angioma serpiginosum should be considered in the diagnosis of this case.

Dr. Irvine recalled the patient shown at Ann Arbor with the lesions pretty well over the whole body, and the diagnosis was given as angioma serpiginosum; but that was not like this at all. Dr. Wise gave that as his opinion, but no one else agreed to it, however.

Ulcer of Pharynx. (Syphilitic?) Presented by Dr. Gager.

The patient, a woman ,aged 25, white, married, came to the University Dispensary two weeks ago. There was an ulcer of the posterior pharyngeal wall. This ulcer was about 3 cm. long and from 1 to 1.5 cm. in width. There was no discharge from the ulcer and very little pus. Smears were made and stained for various organisms, including Vincent's spirillum, and the laboratory findings were negative. The Wassermann test of the serum was negative.

The diagnosis of syphilitic ulceration was made and the patient was given neo-arsphenamin, 0.6 gm., one week ago and a second dose today. The first dose of neo-arsphenamin given one week ago did not seem to have made the impression on the ulcer that usually follows. There was not the local reaction that one usually sees. In addition, the patient gave the history of having fallen and bruised her leg seven weeks ago and still has a periostitis. A roentgenogram of the leg, taken at that time, was negative.

A diagnosis of tuberculous ulcer would have to be considered in this case, but a therapeutic test should rule out the diagnosis of syphilis.

DISCUSSION

Dr. Armstrong said he was unable to make a diagnosis in this case. He also wished to state that he thought it a mistake to give arsphenamin in these doubtful cases. He realized, of course, that a therapeutic diagnosis can be

made in syphilis, but he thought it a mistake to try it in doubtful ones. Potassium iodid can be used just as well and he did not like to subject this type of patients to arsphenamin.

In answer to Dr. Cook's question as to why he did not approve, Dr. Armstrong stated that, for one thing, he had a couple of patients die from the effects of arsphenamin and he did not want any more.

Dr. Gager explained in this connection that his first diagnosis of the case a week ago was syphilis; and the fact that the patient had had only one dose, even if she has not had the reaction that one often sees, does not rule out syphilis. He made the reservation now, and he felt he would not be so positive in that diagnosis now without the positive test. He thought Dr. Armstrong's point was well taken, and felt that Dr. Armstrong was justified in saying that unless we can make a diagnosis of syphilis we should not subject patients to arsphenamin. One often does see arsphenamin dermatitis, which is very serious to the patient. He felt also that we should not be too hasty in our use of arsphenamin in these cases.

Dr. Irvine stated that this was a very interesting case and represented a problem we often meet.

Dr. Michelson said that he could not see any distinct ulceration. The patient had had a tonsillectomy performed and there were bits of lymphatic tissue still remaining. Although the Wassermann test was positive on one occasion, clinically she was completely normal. Her husband was living and well, and she had two children living and well. There had been no miscarriages. Because of irregularity of the tibia a roentgenogram had been made which was reported not syphilitic. Dr. Michelson's diagnosis was traumatic change of the pillars following surgery.

Dr. Goeckerman stated that he would not venture to make any diagnosis in this case. In connection with the use of arsphenamin in this type of case, Dr. Goeckerman stated that he would like to call attention to something that has been called to our attention before; i. e., that the use of arsphenamin does not constitute a final therapeutic test. They had seen tuberculous ulcers heal under the use of arsphenamin. He stated that his own best guess, not knowing any more about the case, would be tuberculous ulceration of the throat.

Dr. Michelson thought, in relation to Dr. Goeckerman's discussion, that one should be more specific than merely making a diagnosis of tuberculosis, for the prognosis depended entirely on what type of tuberculosis of the skin or mucous membrane we were dealing with. Tuberculous ulceration of the tongue or pharynx usually occurred in persons with advanced pulmonary tuberculosis. These ulcers have have the appearance of a chancroid. The base is studded with grayish spots. There is a rapid cheesy degeneration, and the edges are distinctly undermined. There is usually quite a copious discharge. The prognosis in these cases is bad. These patients often react negatively to tuberculin. Lupus vulgaris, on the other hand, is a low grade tuberculosis, and is characterized by the formation of lupus nodules even though ulceration is present; lupus nodules are essential for such a diagnosis. Dr. Michelson thought it was very difficult to diagnose lupus vulgaris on the mucous membranes when there is no lesion on the skin. The prognosis, as is well known, is good in lupus vulgaris. Usually lupus vulgaris made an effort to heal, scar tissue readily forming, while in the ulcerating form healing was never seen. Another form of tuberculosis of the tongue, which is exceedingly rare,

is the so-called tuberculoma, which is similar to scrofuloderma gummosa. It is a tuberculous process occurring subepithelially, making a node. There is an illustration of such a case in Leinandowsky's book.

Dr. Goeckerman stated that his conception of tuberculosis of the mucous membranes leads him to feel that this need not be anything but lupus vulgaris, and yet not have microscopic evidence tending to confirm a diagnosis of tuberculosis; he felt that in this case it could not be excluded without further study of the case.

Dr. Butler believed that it was definitely a tuberculous ulceration; and while it is true, as Dr. Michelson has stated, that tuberculous ulceration in such a location is almost always seen with systemic tuberculosis, he had seen such tuberculous ulceration without systemic tuberculosis. He would certainly rule out lupus vulgaris as, while there have been cases of lupus beginning after the age of puberty, he did not believe in the world's history you could pick out ten cases beginning in adult life.

Dr. Armstrong stated that he had no history in this case. He thought it quite possible that an accurate history might change the diagnostic point of view. He would not make a diagnosis of syphilis.

Dr. Sweitzer said it did not look particularly syphilitic to him. He would hesitate to make a diagnosis of tuberculosis. He recalled that Dr. F. Harris showed a case of tuberculosis of the cheek in Chicago a number of years ago. Some thought it was carcinoma, but Dr. Harris had diagnosed it as tuberculosis. He believed that was painful. Section showed it to be tuberculosis. Dr. Sweitzer suggested that if the patient did not improve, he should be advised to see a nose and throat specialist and have him take out a section for examination. The findings might help a good deal in arriving at a diagnosis.

Dr. Irvine said that if such lesions are syphilitic they are quite destructive. This is a matter of great importance to the patient, for waiting for the physician to decide, often a great deal of tissue is destroyed. He asked for a further report on this case at a later meeting.

D. D. Turnacliff, M.D., Secretary.

PHILADELPHIA DERMATOLOGICAL SOCIETY

Regular Monthly Meeting, Feb. 11, 1924

Fred D. Weidman, M.D., *Presiding*

Scleroderma and Sclerodactylia. Presented by Dr. Weidman.

Ethel J., colored, aged 37, had been regarded in the hospital wards as having a case of arthritis deformans. The usual distribution over the cheeks, hands and feet was observed, as well as the thinning and hide-binding, and stiffening of the phalangeal, tarsal and carpal joints. All digits were markedly deformed and twisted and the distal ones retroflexed. The superficial parts of the skin were comparatively normal. The disease was affecting the deeper parts, and more certainly the subcutaneous tissues were sclerosed, and apparently the bones were atrophic. This was probably one of the cases of deep scleroderma—peri-articular pansclerosis, extending well below the subcutaneous parts. The

mental process of the mandible was so atrophic that the patient's chin had a froglike appearance. It was believed that the patient was improving on thyroid extract. Records showed inability to obtain blood for examination.

DISCUSSION

DR. SCHAMBERG said he thought that the percentage of patients with scleroderma that react in a material way to thyroid medication was small, although in some cases improvement almost to the extent of cure had been recorded, and there were others in which the combined use of pituitary and thyroid gland medication had given good results.

ARSENICAL PIGMENTATION AND KERATOSIS. Presented by DR. WEIDMAN.

John R., a white adult, had an anemia of progressive, pernicious type, probably secondary to abdominal neoplasm, and had received heavy arsenical medication for more than a year and a half. The face was free from pigmentation, but elsewhere the skin was diffusely brown; the color was particularly marked on the loins and on the thighs. The keratosis was diffuse and superficial. The arsenical treatment had been discontinued during the last week, and much of the pigmented skin had exfoliated in the form of a branny scale, leaving a less strongly pigmented skin underneath.

DISCUSSION

DR. SCHAMBERG said he thought the condition was partly due to the arsenic, but that there was a condition characterized by scaliness and pigmentation of the skin that occurred in patients who were ill from some internal or systemic disorder.

RECKLINGHAUSEN'S DISEASE WITH PIGMENTATION AROUND OPERATIVE SCARS. Presented by DR. WEIDMAN.

H. B., a woman, white, aged 50, came to the hospital complaining of dysphagia, but esophagoscopic and fluoroscopic examinations were negative. A lipoma was said to have been removed from the urinary bladder, following which the tumors and freckles developed. The numerous cavernous telangiectasis of the face had been present prior to operation. None of the tumors at this time was larger than a bean, but four of the large ones had been removed surgically; around the scars of each there was a diffuse pigmentation which was considered to be due rather to local irritation than to the usual processes in the disease.

NEUROFIBROMA. Presented by DR. GREENBAUM.

A man had a tumor removed from the forearm, and later developed small tumors over the body, associated with numerous pigmented patches.

DISCUSSION OF THE TWO PRECEDING CASES

DR. WEIDMAN said he was interested in the character of the pigmentation surrounding the scar tissue, and spoke of conditions due to foci of irritation and increased transmission of nervous impulses near scars.

DR. SCHAMBERG said he considered the etiology the important point in these cases, and suggested a thorough study along the line of the ductless glands.

Dr. Lavinder recalled two cases of Recklinghausen's disease in which biopsy had been done, and in one of the cases pigmentation followed at the site of the biopsy, while no pigmentation appeared in the other case.

Extensive Keloid of Face Following Burn. Presented by Dr. Pfahler.

A girl, aged 11, presented keloid of the face following a burn; she had been treated by roentgen ray, with satisfactory results, but considerable thickening still existed in the nasolabial folds. Dr. Pfahler desired the opinion of the members of the Society as to whether this part of the face should be treated surgically or whether the roentgen-ray treatment should be continued.

DISCUSSION

Dr. Schamberg advised a continuation of the use of the roentgen ray, in view of the excellent results that had been obtained. He said that he did not believe that these keloids developing in children following a burn showed any particular keloid tendency on the part of the patient.

A Case for Diagnosis. Presented by Dr. Wright.

One year ago this patient was seen by Drs. Schamberg and Wright, at which time the nose presented over its entire surface a crusted lesion. These crusts were removed, exposing beneath an exuding, acutely inflammatory surface showing numerous dilated follicular openings. The undersurfaces of the crusts were studded with numerous stalactite-like projections which had fitted into the follicular openings, making the crusts adherent. The patient gave a history of having received roentgen-ray treatments, which had aggravated the condition. A tentative diagnosis of eczema with roentgen-ray dermatitis was made, and soothing remedies applied, but without benefit. Later the diagnosis was changed to acute lupus erythematosus, and following applications of carbon dioxid snow the nose healed, and the patient disappeared from observation. Three days ago, he presented himself at the Polyclinic Hospital, and examination revealed a recurrence of the eruption on the nose with extension on to the cheeks. Numerous bullous lesions were present on the lips and over the upper part of the chest and shoulders. The condition now is suggestive of a pemphigus.

DISCUSSION

Dr. Weidman said he considered this case tentatively as a bleb form of erythema multiforme.

Dr. Klauder said he thought it was a case of lupus erythematosis.

Dr. Greenbaum considered it a case of pemphigus.

Dr. Schamberg said that originally the eruption on the face looked like a lupus erythematosis, but the subsequent development of bullae in the mouth and on the chest showed it to be a case of pemphigus.

Monilethrix. Presented by Drs. E. F. Corson and Patricia Hart-Drant.

Hairs taken from different portions of the scalp of a white child, aged 5, were shown under the microscope. The disease had been first noticed at the age of 6 months. There was no history of other members of the family exhibiting the same condition. There was much thinning and fragility of the hair, and a typical succession of spindles along the shaft was noted under the low power microscope.

Patricia Hart-Drant, Secretary.

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Antisyphilitic Treatment, Kidneys and. L. Lartat-Jacob, Médecine **5**:444 (March) 1924.

Aortic Aneurysm, Roentgen-Ray Diagnosis of. E. C. Samuel, Am. J. Roentgenol. **11**:361 (April) 1924.

Aortic Pain and Antisyphilitic Treatment. C. G. Cumston, Am. J. Syphilis **8**:349 (April) 1924.

Arsenic, Bismuth and Mercury Poisoning, Antidote for. H. C. Semon, Brit. M. J. **1**:662 (April 12) 1924.

Arsenic Content of Blood After Intravenous Injection of Tryparsamide. XII. J. A. Fordyce, I. Rosen and C. N. Myers, Am. J. Syphilis **8**:264 (April) 1924.

Arsenic Content of Cerebrospinal Fluid After Intravenous Injection of Hypertonic Saline Solution and Neo-Arsphenamin. J. A. Fordyce, I. Rosen and C. N. Myers, Am. J. Syphilis **8**:297 (April) 1924.

Arsenic in Serum and Clot After Intravenous Administration of Arsphenamin, Neo-Arsphenamin and Silver Arsphenamin. Quantitative Studies in Syphilis from Clinical and Biologic Point of View. XI. Partition of. J. A. Fordyce, I. Rosen and C. N. Myers, Am. J. Syphilis **8**:193 (April) 1924.

Arsphenamin and Blood Coagulability. G. H. Copher, J. A. M. A. **82**:1571 (May 10) 1924.

Arsphenamin, "Cure" of Syphilis in Rabbit with. A. M. Chesney and J. E. Kemp, J. Exper. Med. **39**:553 (April) 1924.

Arsphenamin Injuries. A. M. v. Werthern, Klin. Wchnschr. **3**:627 (April 8) 1924.

Arsphenamin and the Liver. A. Kartamischew, Arch. f. dermat. u. Syph. **147**: 100 (April 24) 1924.

Arsphenamin with Methyl Violet, Increase of Permeability of Choroid Plexus to. D. C. Smith and J. A. Waddell, Am. J. Syphilis **8**:310 (April) 1924.

Arsphenamin-Resistant Syphilis. S. Silberstein, Arch. f. dermat. u. Syph. **147**:116 (April 24) 1924.

Arsphenamin, Standardization of. De Myttenacre, Bull. de l'Acad. roy de Méd. Brussels **4**:65 (Feb. 23) 1924.

Bismuth Compounds in Treatment of Syphilis. Fournier et al., Ann. de l'Inst. Pasteur **38**:240 (March) 1924.

Bismuth in Congenital Syphilis. M. Frank, Arch. f. Kinderh. **74**:106 (April 5) 1924.

Bismuth and Mercury Poisoning, Antidote for Arsenic. H. C. Semon, Brit. M. J. **1**:662 (April 12) 1924.

Bismuth in Treatment of Syphilis. Hudelo and Rabut, Presse méd. **32**:313 (April 9) 1924.

Bismuth in Trypanosomiasis and Spirillosis. C. Levaditi and S. Nicolau, Ann. de l'Inst. Pasteur **38**:179 (March) 1924.

Blood Coagulability, Arsphenamin and. G. H. Copher, J. A. M. A. **82**:1571 (May 10) 1924.

Cardiovascular Disease, Etiology of. N. P. Barnes, J. A. M. A. **82**:1571 (May 10) 1924.

Cerebral Syphilis, Unusual Eye Manifestations of Case. C. B. Welton, Illinois M. J. **15**:274 (April) 1924.

Cerebral Ventricles in Syphilis. Cestan et al., Ann. de méd. **15**:201 (March) 1924.

Cerebrospinal Fluid After Intravenous Injection of Hypertonic Saline Solution and Neo-Arsphenamin, Arsenic Content of. XIII. J. A. Fordyce, I. Rosen and C. N. Myers, Am. J. Syphilis **8**:297 (April) 1924.

Chancre in Juvenile with General Paralysis. Clyde E. Shinkle, J. A. M. A. **82**:1780 (May 31) 1924.

Cholesterin Reinforced Antigen in Wassermann Reaction, Apparent Specific Action of. E. S. Gault, J. Lab. & Clin. Med. **9**:486 (April) 1924.

Choroid Plexus to Arsphenamin with Methyl Violet, Increase of Permeability of. D. C. Smith and J. A. Waddell, Am. J. Syphilis **8**:310 (April) 1924.

Colloidal Gold Reaction. E. Bloch and H. Biberfeld, Deutsche med. Wchnschr. **50**:461 (April 11) 1924.

Colloids, Seriodiagnosis of Syphilis and. R. Stern, J. A. M. A. **82**:1575 (May 10) 1924.

Complement Fixation for Diagnosis of Syphilis, Kolmer Method of. S. H. Kellogg, A. Wells and D. Beck, J. Lab. & Clin. Med. **9**:481 (April) 1924.

Complement Fixation Test for Syphilis Specificity and Sensitiveness of, Author's New. J. A. Kolmer, J. Lab. & Clin. Med. **9**:468 (April) 1924.

Complement Fixation, Theory of. M. Renaud, Compt. Rend. Soc. de biol. **90**:741 (March 28) 1924.

Congenital Syphilis. G. Nobl and F. Remenovsky, Wien. klin. Wchnschr. **37**:341 (April 3) 1924.

Congenital Syphilis in Bismuth. M. Frank, Arch. f. Kinderh. **74**:106 (April 5) 1924.

Congenital Syphilis, Oculomotor Palsy with Parenchymatous Keratitis in. A. F. Luhr, Am. J. Ophth. **7**:293 (April) 1924.

Congenital Syphilis, Prophylaxis of. J. Almkvist, Hygiea **86**:113 (March 15) 1924.

Congenital Syphilis, Treatment of. K. Italiener, Klin. Wchnschr. **3**:577 (April 1) 1924.

Diagnosis of Syphilis, Kolmer Method of Complement Fixation for. S. H. Kellogg, A. Wells and D. Beck, J. Lab. & Clin. Med. **9**:481 (April) 1924.

Early Syphilis, Lumbar Puncture in. A. Goldscheider and A. Wittgenstein, Deutsch. med. Wchnschr. **50**:508 (April 18) 1924.

Effect on Kidney of Treatment for Syphilis. A. H. McFarland, Am. J. M. Sc. **176**:477 (April) 1924.

Eye Manifestations of Cerebral Syphilis. Unusual Case. C. B. Welton, Illinois M. J. **15**:274 (April) 1924.

Flocculation Reaction. A. Dalla Volta and P. Benedetti, Policlinico **31**:189 (April 1) 1924.

Flocculation Reaction as Substitute for Wassermann Test. F. Green, J. Lab. & Clin. Med. **9**:447 (April) 1924.

Gastro-Intestinal Hemorrhage in, Tabes. S. Poliakoff, J. A. M. A. **82**:1576 (May 10) 1924.

General Paralysis, with Chancre in Juvenile. Clyde E. Shinkle, J. A. M. A. **82**:1780 (May 31) 1924.

General Paralysis, of Hemolysins in Pathogenesis. V. Kafka, Med. Klin. **20**:456 (April 6) 1924.

General Paralysis, Latent Period of. F. Wiesel, Hygiea **86**:164 (March 15) 1924.

General Paralysis, Treatment of. A. Starcke, Nederlandsch Tidjschr. v. Geneesk. **1**:1751 (April 12) 1924.

General Paralysis, Treatment of Malaria. G. Herrmann, Med. Klin. **20**:445 (April 6) 1924.

Gumma on Humerus and Pseudoparalysis. G. Mouriquand and M. Bernheim, Arch. de méd. d'enf. **27**:228 (April) 1924.

Hemolysins in Pathogenesis of General Paralysis. V. Kafka, Med. Klin. **20**:456 (April 6) 1924.

Hemorrhages of Infancy and Childhood. A. F. G. Spinks, Brit. M. J. **1**:625 (April 5) 1924.

Hydrocele in Infant with Inherited Syphilis. G. Sales and P. Vallery-Radot, Nourrisson **12**:138 (March) 1924.

Inherited Syphilis, Hydrocele in Infant with. G. Sales and P. Vallery-Radot, Nourrisson **12**:138 (March) 1924.

Injection of Hypertonic Saline Solution and Neo-Arsphenamin, Arsenic Content of Cerebrospinal Fluid After Intravenous. XIII. J. A. Fordyce, I. Rosen and C. N. Myers, Am. J. Syphilis **8**:297 (April) 1924.

Intravenous Administration of Arsphenamin, Neo-Arsphenamin and Silver Arsphenamin. Quantitative Studies in Syphilis from Clinical and Biologic Point of View. XI. Partition of Arsenic in Serum and Clot After. J. A. Fordyce, I. Rosen and C. N. Myers, Am. J. Syphilis **8**:193 (April) 1924.

Kahn Reaction in Serologic Diagnosis of Syphilis. H. L. Keim, Am. J. Syphilis **8**:323 (April) 1924.

Kahn and Wassermann Tests on 500 Serums, Comparison of. T. E. Osmond and D. McClean, Brit. M. J. **1**:617 (April 5) 1924.

Kidney, Effect on, of Treatment for Syphilis. A. R. McFarland, Am. J. M. Sc. **176**:477 (April) 1924.

Kolmer Method of Complement Fixation for Diagnosis of Syphilis. S. H. Kellogg, A. Wells and D. Beck, J. Lab. & Clin. Med. **9**:481 (April) 1924.

Kolmer Method and Routine Wassermann Technic on Fourteen Hundred Tests, Comparative Results of. M. Cohen and S. R. Haythorn, J. Lab. & Clin. Med. **9**:476 (April) 1924.

Liver, Arsphenamin and the. A. Kartamischew, Arch. f. dermat. u. Syph. **147**: 100 (April 24) 1924.

Lumbar Puncture in Early Syphilis. A. Goldscheider and A. Wittgenstein, Deutsch. med. Wchnschr. **50**:508 (April 18) 1924.

Malaria Treatment of General Paralysis. G. Herrmann, Med. Klin. **20**:445 (April 6) 1924.

Meinicke Test, Simplified. R. Untersteiner, Deutsch. med. Wchnschr. **50**:405 (March 28) 1924.

Meinicke's Turbidity Reaction. M. Richter, Deutsch. med. Wchnschr. **50**:436 (April 4) 1924.

Meinicke's Turbidity Reaction. W. Schultze, Klin. Wchnschr. **3**:580 (April 1) 1924.

Mercury Poisoning, Antidote for Arsenic, Bismuth and. H. C. Semon, Brit. M. J. **1**:662 (April 12) 1924.

Methyl Violet, Increase of Permeability of Choroid Plexus to Arsphenamin with. D. C. Smith and J. A. Waddell, Am. J. Syphilis **8**:310 (April) 1924.

Neo-Arsphenamin and Silver Arsphenamin. Quantitative Studies in Syphilis from Clinical and Biologic Point of View. XI. Partition of Arsenic in Serum and Clot After Intravenous Administration of Arsphenamin. J. A. Fordyce, I. Rosen and C. N. Myers, Am. J. Syphilis **8**:193 (April) 1924.

Oculomotor Palsy with Parenchmatous Keratitis in Congenital Syphilis. A. F. Luhr, Am. J. Ophth. **7**:293 (April) 1924.

Ophthalmoplegia in Syphilitic Basilar Meningitis. A. H. Pember, Am. J. Ophth. **7**:294 (April) 1924.

Osteitis, Syphilitic of the Skull. A. Leri and P. Cottenot, Bull. de la Soc. méd. d. hôp., Paris **48**:432 (March 28) 1924.

Parenchymatous Keratitis in Congenital Syphilis, Oculomotor Palsy with A. F. Luhr, Am. J. Ophth. **7**:293 (April) 1924.

Pituitary Body, Syphilis of Vocal Cords and Spinal Cord. R. A. Hernandez, Am. J. Syphilis **8**:321 (April) 1924.

Porokeratosis Primarily Affecting Glans Penis and Mucous Membrane of Mouth, Case of So-Called. H. Yanagihara and K. Sakamoto, J. Oriental Med. **2**:67 (Feb.) 1924.

Precipitation Test for Syphilis as Public Health Laboratory Procedure. L. C. Haven and M. L. Taylor, Am. J. Pub. Health **14**:323 (April) 1924.

Pseudoleukemia, Syphilitic. A. Nanta, Médecine **5**:467 (March) 1924.

Quantitative Studies in Syphilis from Clinical and Biologic Point of View. XI. Partition of Arsenic in Serum and Clot After Intravenous Administration of Arsphenamin, Neo-Arsphenamin and Silver Arsphenamin. J. A. Fordyce, I. Rosen and C. N. Myers, Am. J. Syphilis **8**:193 (April) 1924.

Reinfection in Syphilis, M. A. Reasoner, Am. J. Syphilis **8**:313 (April) 1924.

Routine Wassermann Technic on Fourteen Hundred Tests, Comparative Results of Kolmer Method and. M. Cohen and S. R. Haythorn, J. Lab. & Clin. Med. **9**:476 (April) 1924.

Sachs-Georgi Flocculation Test for Syphilis; Simplification of Technic. S. Hata, Am. J. Syphilis **8**:334 (April) 1924.

Serodiagnosis, Quantitative of Syphilis. F. F. Werner, Deutsch. med. Wchnschr. **50**:405 (March 28) 1924.

Serodiagnosis, of Syphilis and Colloids. R. Stern, J. A. M. A. **82**:1575 (May 10) 1924.

Serologic Diagnosis of Syphilis, Kahn Reaction in. H. L. Keim, Am. J. Syphilis **8**:323 (April) 1924.

Silver Arsphenamin, Quantitative Studies in Syphilis from Clinical and Biologic Point of View. XI. Partition of Arsenic in Serum and Clot After Intravenous Administration of Arsphenamin, Neo-Arsphenamin. J. A. Fordyce, I. Rosen and C. N. Myers, Am. J. Syphilis **8**:193 (April) 1924.

Spinal Cord, Syphilis of Vocal Cords, Pituitary Body and Spinal Cord. R. A. Hernandez, Am. J. Syphilis **8**:321 (April) 1924.

Spirillosis, Bismuth in Trypanosomiasis and. C. Lebaditi and S. Nicolau, Ann. de l'Inst. Pasteur **38**:179 (March) 1924.

Standardization of Arsphenamin. De Myttenacre. Bull. de l'Acad. roy. de Med. Brussels **4**:65 (Feb. 23) 1924.

St. Paul's Bay, Syphilis Disease at. W. R. Riddell, Pub Health J. **15**:145 (April) 1924.

Sulpharsphenamin, Treatment with. J. Fabry, Med. Klin. **20**:486 (April 13) 1924.

Syphilis, Abortive Treatment of. R. Mutschler, Arch. f. dermat. u. Syph. **147**: 107 (April 24) 1924.

Syphilis of Central Nervous System and of Aorta. K. Lowenberg, Klin. Wchnschr. **3**:531 (March 25) 1924.

Syphilis, the Cerebral Ventricles in. Cestan et al., Ann. de méd. **15**:201 (March) 1924.

Syphilis from Clinical and Biologic Point of View. XI. Partition of Arsenic in Serum and Clot After Intravenous Administration of Arsphenamin, Neo-Arsphenamin and Silver Arsphenamin. Quantitative Studies in. J. A. Fordyce, I. Rosen and C. N. Myers, Am. J. Syphilis **8**:193 (April) 1924.

Syphilis, Disease at St. Paul's Bay. W. R. Riddell, Pub. Health J. **15**:145 (April) 1924.

Syphilis, as Public Health Laboratory Procedure, Precipitation Test for. L. C. Havens and M. L. Taylor, Am. J. Pub. Health **14**:323 (April) 1924.

Syphilis, Quantitative Serodiagnosis of. F. F. Werner, Deutsch. med. Wchnschr. **50**:405 (March 28) 1924.

Syphilis in Rabbit with Arsphenamin, "Cure" of. A. M. Chesney and J. E. Kemp, J. Exper. Med. **39**:553 (April) 1924.

Syphilis, Reinfection in. M. A. Reasoner, Am. J. Syphilis **8**:313 (April) 1924.

Syphilis, Serodiagnosis of, and Colloids. R. Stern, J. A. M. A. **82**:1575 (May 10) 1924.

Syphilis, Specificity and Sensitiveness of Author's New Complement Fixation Test for. J. A. Kolmer, J. Lab. & Clin. Med. **9**:468 (April) 1924.

Syphilis, Treatment of Bismuth in. Hudelo and Rabut, Presse méd. **32**:313 (April 9) 1924.

Syphilis, Treatment of Late. G. A. Dix, Boston M. & S. J. **190**:742 (May 1) 1924.
Syphilis of Vocal Cords, Pituitary Body and Spinal Cord. R. A. Hernandez, Am. J. Syphilis **8**:321 (April) 1924.

Tabes, Gastro-Intestinal Hemorrhage in. S. Poliakoff, J. A. M. A. **82**:1576 (May 10) 1924.
Tabes, The Waves of Aggravation in the Evolution of. C. Foiz and H. La Grange, Bull. méd., Paris **38**:375 (April 5) 1924.
Test for Syphilis, Complement Fixation Specificity and Sensitiveness of Author, New. J. A. Kolmer, J. Lab. & Clin. Med. **9**:468 (April) 1924.
Treatment of Syphilis, Bismuth Compounds in. Fournier et al., Ann. de l'Inst. Pasteur **38**:240 (March) 1924.
Treatment of Late Syphilis. G. A. Dix, Boston M. & S. J. **190**:742 (May 1) 1924.
Treatment for Syphilis, Effect on Kidney. A. H. McFarland, Am. J. M. Sc. **176**:477 (April) 1924.
Treatment of Syphilis, Pooled Arsphenaminized Fortified Serum in. J. G. Marthens, Am. J. Syphilis **8**:349 (April) 1924.
Trigeminal Neuralgia; Avulsion of Sensory Root. Major. C. W. Flynn, Texas State J. M. **19**:677 (April) 1924.

Venereal Diseases, Law on. F. Pinkus, Med. Klin. **20**:472 (April 6) 1924.
Vocal Cords, Syphilis of Pituitary Body and Spinal Cord. R. A. Hernandez, Am. J. Syphilis **8**:321 (April) 1924.

Wassermann and Meinicke Reactions, Comparative Study of. C. B. McGlumphy, J. Lab. & Clin. Med. **9**:453 (April) 1924.
Wassermann Reaction, Apparent Specific Action of Cholesterin Reinforced Antigen in. E. S. Gault, J. Lab. & Clin. Med. **9**:486 (April) 1924.
Wassermann Reaction, Reactivation of the. G. Spremolla, Policlinico **31**:214 (April 1) 1924.

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Abscesses in Axilla, Vaccine Treatment of. G. Duttmann, Med. Klin. **20**:479 (April 13) 1924.
Absorption of Toxin of Bacillus Botul Through Fresh Wounds and from Mucous Surfaces, Danger of. J. C. Geiger, Am. J. Pub. Health **14**:309 (April) 1924.
Acidosis, The Use of Insulin in a Case of, Following Trauma. G. G. Ginsberg, J. A. M. A. **82**:1517 (May 10) 1924.
Adipsogenitalis, Dystrophia. H. Schack, Deutsch. med. Wchnschr. **50**:434 (April 4) 1924.
Allergy and Clinical Picture of Smallpox. Einsiedel, Med. Klin. **20**:441 (April 6) 1924.
Anaphylaxis, Fatal, After Hemoplastin. J. A. M. A. **82**:1564 (May 10) 1924.
Anemia, Present Status of Treatment of. Klieneberger, Deutsch. med. Wchnschr. **50**:523 (April 18) 1924.
Anthrax, Chronic or Retarded. S. Costa et al., Compt. rend. Soc. de biol. **90**:859 (April 4) 1924.
Anthrax, Intradermal Vaccination Against. H. Velu, Compt. rend. Soc. de biol. **90**:746 (March 28) 1924.
Anthrax, Pulmonary Infection with. D. Combiesco, Compt. rend. Soc. de biol. **90**:752 (March 28) 1924.
Anthrax, Vaccination of Horse Against. Brocq-Rousseu and Urbain, Ann. de l'Inst. Pasteur **38**:268 (March) 1924.
Anthrax in White Rats. I. Hababousala, Compt. rend. Soc. de biol. **90**:849 (April 4) 1924.
Arc Lamps, New, for Medical Use. H. Moldenschardt, Deutsch. med. Wchnschr. **50**:410 (March 28) 1924.
Arthritis Deformans, Heart in. Ernst P. Boas and Philip Rifkin, J. A. M. A. **82**:1596 (May 17) 1924.
Autogenous Vaccines, Experiences with. Tietz, Deutsch. med. Wchnschr. **50**: 432 (April 4) 1924.

Bacillus Botulinus, Through Fresh Wounds and from Mucous Surfaces, Danger of Absorption of Toxin. J. C. Geiger, Am. J. Pub. Health **14**:309 (April) 1924.

Bilirubin Formation and Jaundice. Bock, Klin. Wchnschr. **3**:638 (April 8) 1924.

Blastomycosis as Complication of Industrial Accidents. R. M. Carter, J. Industrial Hygiene **5**:457 (April) 1924.

Blue Nevus. H. Stranz, Arch. f. dermat. u. Syph. **147**:131 (April 24) 1924.

Breast Tissues: Hyperplasia of, Mazoplasia. G. L. Cheatle, Brit. M. J. **1**:653 (April 12) 1924.

Bromid Eruption Due to Bromo-Seltzer. J. Frank Waugh, J. A. M. A. **82**:1584 (May 17) 1924.

Bromo-Seltzer, Bromid Eruption Due to. J. Frank Waugh, J. A. M. A. **82**:1584 (May 17) 1924.

Cancer of Breast, Management of. A. W. Erskine, J. Iowa M. Soc. **14**:158 (April) 1924.

Cancer Producing Factor in Tar Cells, Metabolism of. E. Wiechmann, J. A. M. A. **82**:1575 (May 10) 1924.

Carbon Monoxid Poisoning, Skin Gangrene After. Idem. Bull. et mém. Soc. méd. d. hôp. de Paris **48**:486 (April 4) 1924.

Cecal Spirochetosis. G. Delamare et al., Bull. de l'Acad. de méd., Par. **91**:462 (April 8) 1924.

Chemotherapy of Rhinoscleroma. V. Jindra, Casop. lek. cesk. **63**:558 (April 5) 1924.

Chilblains, Roentgen-Ray Treatment of. F. Kroll, Med. Klin. **20**:485 (April 13) 1924.

Chronic Purpura, Treatment of. A. Ricaldoni and Albo, Bull. et mém. Soc. méd. d. hôp. de Paris **48**:486 (April 4) 1924.

Congenital Lymphangioma with Secondary Giantism. Willemse, Nederlandsch Tijdschr. v. Geneesk. **1**:1755 (April 12) 1924.

Diphtheria, Wound. W. Grossmann, Beitr. z. klin. Chir. **131**:461, 1924.

Dystrophia Adiposogenitalis. H. Schack, Deutsch. med. Wchnschr. **50**:434 (April 4) 1924.

Embolic Gangrene of Arm, Report of Case. J. H. Moore, Southwestern Med. **8**:163 (April) 1924.

Epidemic Jaundice: Report of Twenty-Eight Cases. W. A. Whitman, Ohio State M. J. **20**:278 (May) 1924.

Epithelioma of Face. W. W. Bowen, J. Iowa M. Soc. **14**:154 (April) 1924.

Epithelioma of Lid and Orbit. Heitor Praguer Froes, Brazil-med. **1**:155 (March 22) 1924.

Erysipeloid. E. v. Redwitz, München. med. Wchnschr. **71**:438 (April 4) 1924.

Erythema Nodosum, Etiology of. G. Lindberg, Hygiea **86**:177 (March 31) 1924.

Genital Carcinomas, Results in Five Hundred. J. Schulte, Arch. f. Gynäk. **121**:4 (March 20) 1924.

Heart in Arthritis Deformans. Ernst P. Boas and Philip Rifkin, J. A. M. A. **82**:1596 (May 17) 1924.

Hemoplastin, Anaphylaxis, Fatal After. J. A. M. A. **82**:1564 (May 10) 1924.

Hemorrhagic Purpura, and Tuberculosis. A. Pratsicas, Grèce méd. **26**:28 (Feb.) 1924.

Henoch's Purpura, Case of. D. Kilgour, Canad. M. A. J. **14**:302 (April) 1924.

Herpes Zoster, Anatomic Lesions in. S. Hesser, Acta Chir. Scandinav. **60**: 278, 1924.

Infectious Disease, Treatment of. L. Saathoff, München. med. Wchnschr. **71**: 392 (March 28) 1924.

Infiltration of Subcutaneous Tissue. G. Wiltrup, Acta. med. Scandinav. **60**:432 (April 15) 1924.

Intradermal Vaccination Against Anthrax. H. Velu, Compt. rend. Soc. de biol. **90**:746 (March 28) 1924.

Jaundice, Bilirubin Formation and. Bock, Klin. Wchnschr. **3**:638 (April 8) 1924.
Jaundice, Familial Hemolytic. H. Hattesen, Mitt. a. Grenzgeb. d. Med. u. Chir. **37**:293, 1924.

Light, Permeability of Skin for. F. Bernhard, München. med. Wchnschr. **71**: 427 (April 4) 1924.
Lupus Pernio, Sarcoid of Boeck and. H. Martenstein, Arch. f. dermat. u. Syph. **147**:70 (April 24) 1924.

Measles, Etiology of. F. Arloing and A. Dufourt, Compt. rend. Soc. de biol. **90**:763 (March 28) 1924.
Measles and Whooping Cough, Voluntary Exposure Dangerous in. H. B. Sears, Wisconsin M. J. **22**:523 (April) 1924.
Mercurial Eruption. E. Hoffert, Arch. f. dermat. u. Syph. **147**:135 (April 24) 1924.
Metabolism of Cancer Producing Factor in tar Cells. E. Wiechmann, J. A. M. A. **82**:1575 (May 10) 1924.
Milk, Transmission of Scarlet Fever by. M. Gioseffi, Riforma méd. **40**:274 (March 24) 1924.
Morphin, Skin Reaction to. J. D. Pilcher and T. Sollmann, Arch. Int. Med. **33**:516 (April) 1924.
Mycetoma, Third Case of. Gustav A. Pagenstecher, J. A. M. A. **82**:1692 (May 24) 1924.

Ophthalmic Herpes Zoster. E. de Campos, Brazil-med. **1**:147 (March 15) 1924.

Pfeiffer Reaction in Yellow Fever, The. J. A. M. A. **82**:1521 (May 10) 1924.
Pigmentation of Bulbar Conjunctiva in Xerpsos Epithelialis Conjunctivae. Pathology of. S. Mori, J. Oriental Med. **2**:103 (Feb.) 1924.
Pointed Condyloma, Warts and. Waelsch and Habermann, Arch. f. dermat. u. Syph. **147**:144 (April 24) 1924.
Pseudoparalysis, Gumma on Humerus and. G. Mouriquand and M. Bernheim, Arch. de méd. d'enf. **27**:228 (April) 1924.

Radiation of Heat by Skin. R. Cobet and F. Bramigk, Deutsch. Arch. f. klin. Med. **144**:45 (April) 1924.
Radiodermatitis. Jeanselme and Giraudeau, Bull. de l'Acad. de méd., Par. **38**:435 (April 19) 1924.
Rheumatism and Erythema Nodosum. R. Anderson, Brit. M. J. **1**:665 (April 12) 1924.
Rhinoscleroma, Chemotherapy of. V. Jindra, Casop. lek. cesk. **63**:558 (April 5) 1924.
Roentgen Rays, Action of Various Antagonistic. A. Dognon, Compt. rend. Soc. de biol. **90**:778 (March 28) 1924.
Roentgen Rays, Tardy Injury from. H. Rahm, Beitr. z. klin. Chir. **131**:456 1924.
Roentgen-Ray Therapy in Higher Voltages. Evolution of. A. Soiland, California & Western Med. **22**:148 (April) 1924.
Roentgen-Ray Treatment of Chilblains. F. Kroll, Med. Klin. **20**:485 (April 13) 1924.

Sarcoid of Boeck and Lupus Pernio. H. Martenstein, Arch. f. dermat. u. Syph. **147**:70 (April 24) 1924.
Scarlet Fever, Etiology of. Z. L. Magni, Riv. di Clin. pediat. **22**:241 (April) 1924.
Scarlet Fever by Milk, Transmission of. M. Gioseffi, Riforma méd. **40**:274 (March 24) 1924.
Scarlet Fever Otitis. E. H. R. Harries and F. B. Gilhespy, Lancet **1**:843 (April 26) 1924.
Scarlet Fever, Reactions in. T. Pontano, Policlinico **31**:219 (April 1) 1924.
Seborrhea of Face After Encephalitis. G. Stiefler, Wien. klin. Wchnschr. **37**: 334 (April 3) 1924.

Skin Changes of Thyroid Origin. Bogroff and Krupnikoff, Arch. f. dermat. u. Syph. **147**:61 (April 24) 1924.
Skin Gangrene After Carbon Monoxid Poisoning. Idem., Bull. et mém. Soc. méd. d. hôp. de Paris **48**:486 (April 4) 1924.
Skin for Light, Permeability of. F. Bernhard, München. Med. Wchnschr. **71**: 427 (April 4) 1924.
Skin, Radiation of Heat by. R. Cobet and F. Bramigk, Deutsch. Arch. f. klin. Med. **144**:45 (April) 1924.
Skin Reaction to Morphin. J. D. Pilcher and T. Sollmann, Arch. Int. Med. **33**:516 (April) 1924.
Skin Tuberculin Reaction, Import of. A. B. Marfan, Nourrisson **12**:73 (March) 1924.
Smallpox, Clinical Picture of Allergy and. Einsiedel, Med. Klin. **20**:441 (April) 1924.
Smallpox Vaccination, History of. H. A. Gins, Klin. Wchnschr. **3**:634 (April 8) 1924.
Sprue, Tropical; Necropsy. A. S. Rosenfeld, Northwest Med. **22**:181 (April) 1924.
Subcutaneous Tissue, Infiltration of. G. Wiltrup, Acta. med. Scandinav. **60**:432 (April 15) 1924.

Tetanus, Treatment and Management of. L. W. Shannon, J. Kansas M. Soc. **24**:95 (April) 1924.
Trauma, The Use of Insulin in a Case of Acidosis Following. G. G. Ginsberg, J. A. M. A. **82**:1517 (May 10) 1924.
Trifacial Neuralgia. A. W. Adson, Illinois M. J. **15**:262 (April) 1924.
Tropical Sprue; Necropsy. A. S. Rosenfeld, Northwest Med. **22**:181 (April) 1924.

Vaccination Has Prevented Nation-Wide Epidemics. H. M. Guilford, Wisconsin M. J. **22**:521 (April) 1924.
Vaccination of Horse Against Anthrax. Brocq-Rousseu and Urbain, Ann. de l'Inst. Pasteur **38**:268 (March) 1924.
Vaccination, Intradermal Against Anthrax. H. Velu, Compt. rend. Soc. de biol. **90**:746 (March 28) 1924.
Vaccine Treatment of Abscesses in Axilla. G. Duttmann, Med. Klin. **20**:479 (April 13) 1924.
Viscerosensory Phenomena, Further Studies on: Acute Cholelithiasis; Acute Nephrolithiasis. E. M. Livingston, J. A. M. A. **82**:1495 (May 10) 1924.

Warts and Pointed Condyloma. Waelsch and Habermann, Arch. f. dermat. u. Syph. **147**:144 (April 24) 1924.
Whooping Cough, Voluntary Exposure Dangerous in Measles and. H. B. Sears, Wisconsin M. J. **22**:523 (April) 1924.
Wounds, Danger of Absorption of Toxin of Bacillus Botulinus. Through Fresh and from Mucous Surfaces. J. C. Geiger, Am. J. Pub. Health **14**:309 (April) 1924.
Wound Diphtheria, W. Grossmann, Beitr. z. klin. Chir. **131**:461, 1924.
Wound Infections, Treatment of. Fessler, Deutsch. med. Wchnschr. **50**:465 (April 11) 1924.
Wounds, Rate of Healing of. J. Brownlee, J. Exper. Med. **39**:603 (April) 1924.
Wounds, Technic of Dressing. Feszler, Deutsch. med. Wchnschr. **50**:406 (March 28) 1924.

Xanthoma Diabeticorum. M. Azer, Brit. M. J. **1**:665 (April 12) 1924.
Xerosis Epithelialis Conjunctivae, Pathology of Pigmentation of Bulbar Conjunctiva in. S. Mori, J. Oriental Med. **2**:103 (Feb.) 1924.

Yellow Fever, The Pfeiffer Reaction in. J. A. M. A. **82**:1521 (May 10) 1924.

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